

ECG Self-Study Book

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ECG

Self-Study Book

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Foreword

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ECG Self-Study Book

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Dedicated to

Late Henry JL Marriott

Whom I greatly respected. We lost a one-of-a-kind true giant in the field of ECG. I particularly enjoyed attending his ECG sessions in the exhibition halls during intermissions of American Heart Association or American College of Cardiology Annual Scientific Conventions (some of you will remember). We became close friends, publishing several articles together. When I was preparing for the publication of the Atlas of ECG, I sat with him side by side on a sofa at his home, going over each page. He encouraged me by saying he liked my approach to ECG. After reading my description, he would often say “this is Wangflair.” May he rest in peace after a glorious career.

My beloved parents

Without whom I would not be here. I was born in South Korea. When the Korean War broke out, my hometown fell into the north. Thinking that the conflict would not last more than three months, my parents persuaded me to take refuge in south alone, which I did. I was 16 years old then. We are still divided 60 years later, a tragic by-product of the cold war. In the meantime, both of them passed away in my absence. Apparently, I was a sickly baby. They would be very proud of me if they knew how I am doing. May they also rest in peace.

FOREWORD

In medicine, many things evolve while a few others remain unchallenged despite the tests of time. Current medical practitioners rely on an array of new tools to supplement the physical examination, and their personal experience. These tools, mostly the result of impressive technological innovation, astonish us with insights into the evolution, localization and severity of cardiovascular disease; insights undreamed of only a few years ago. Nevertheless, certain elements of our toolkit remain essential foundations of patient care; perhaps the most important is the medical history, but the electrocardiogram (ECG) remains not far behind.

Without conscientious attention paid to medical history-taking, an irreplaceable tool, the relevance of technology's offerings to the care of the individual cannot be placed in proper perspective. Absent perspective we may err by focusing on conditions that are not relevant, and create adverse consequences that are unwarranted. Similarly, the electrocardiogram (ECG) offers a unique perspective; one that is not obtainable by other means. The ECG informs us of an aspect of the patient's health that is often remediable given proper understanding of the message. Whereas the physical examination with the help of various imaging techniques unmasks the impact of disease on the heart's mechanical function, only the ECG informs us of the heart's crucial electrical health.

Since the first half of the 20th century, the ECG has remained mostly unchanged and its importance unchallenged. Professor K Wang has made a career of studying it, and unmasking its messages. He has earned the reputation of being able to detect subtle ECG findings and make complicated ECG matters simple, logical and easy to understand. His recently published *Atlas of Electrocardiography* (2013) offers a comprehensive overview. This *ECG Self-Study Book* is the perfect accompaniment; one which through its more than 400 practice tracings, reinforces the importance and value of ECG understanding. Together they offer an enjoyable and comprehensive ECG education. The internist, emergency room physician, cardiologist, surgeon and family practitioner cannot be considered fully armed and capable in the absence of this education, and I am delighted to recommend this newest of Professor Wang's diligent exposes of this crucial subject to them.

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PREFACE

ECG is a simple diagnostic tool which provides a wealth of clinically useful information. It is rather timely, after the publication of the book *Atlas of Electrocardiography*, to publish this work, *ECG Self-Study Book*, as a companion to it.

In this self-study book, I compiled over 400 practice tracings representing nearly all clinical entities we commonly encounter. Each page has an ECG tracing for you to analyze. Some have multiple choice questions and some require drawing a ladder diagram. Answers or diagnoses and discussions are in the bottom of the tracing for you to compare with yours. Salient features of the tracing, the logic behind the ECG findings, and the clinical relevance of the findings are emphasized.

The book is divided into 30 sections, each section containing 12 cases. The idea is that if one studies one section each day, one would finish the book in 30 days. Of course, one does not have to follow the schedule.

It is hoped that, after practicing on these tracings, one would feel more comfortable in dealing with ECG. Not only does it becomes easier to pick up subtle findings but also understand why the ECG findings manifest the way they do, what is happening to the patient, and what to do about it. In so doing, one would be better prepared to deliver better patient care, which, after all, is what matters. In so doing, the patient care becomes more enjoyable.

K. Wang MD

ACKNOWLEDGMENTS

I am grateful to my daughter, Leah, for her editorial assistance. I also deeply appreciate the secretarial work of Rosie Robinson, Jennifer Walker, Michelle Page, Esther Almeida, Marissa Weatherhead, Wendy Markuson and Yen Nguyencong who graciously put up with my endless revisions. I also thank Cynthis DeKay for coming up with a fine and elegant design for the book cover. I appreciate M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India, for undertaking the difficult task of publishing this book so that the skills of interpreting ECGs will be improved, which will ultimately translate into better patient care.

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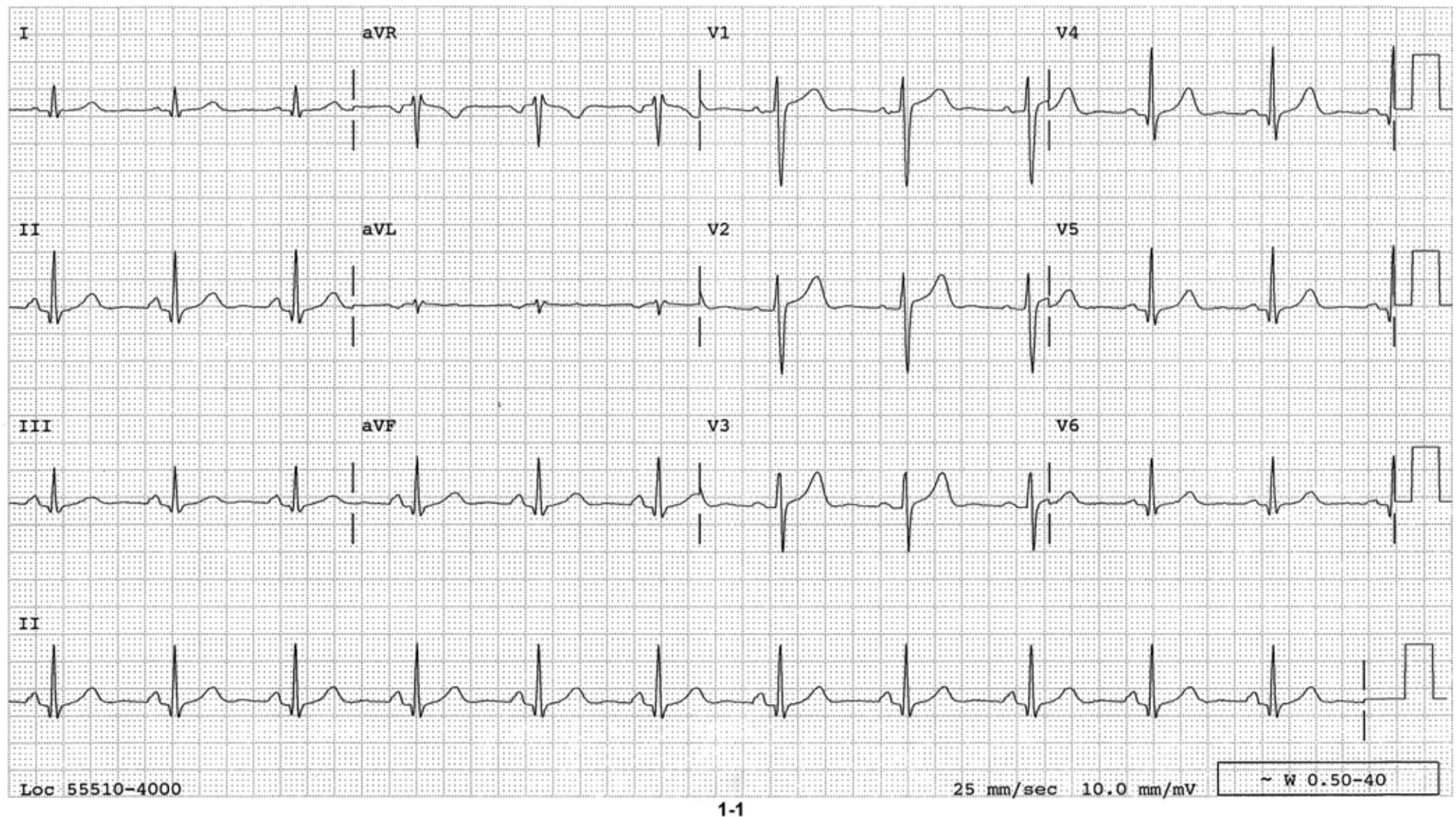
Stress cardiomyopathy. Atrial flutter. Artifact mimicking atrial flutter. 2:1 AV block. Anteroseptal infarction and RBBB. Accelerated idioventricular rhythm with 1:1 VA conduction. Acute pericarditis. Nonconducted PACs causing pauses. Extensive old anterior infarct. Nonconducted atrial premature impulse. 2° AV block, Type I. 2° AV block, Type II. Sinoatrial block, Type II. The changing QRS height due to the timing of the QRS in relationship to the flutter waves. Summation of the P wave and R wave. Rate-dependent BBB. Ventricular bigeminy. Respiration causes the QRS height and configuration to change. Respiratory variation in QRS morphology mimicking EA. Respiratory variation resulting in a gradual increase and decrease in the height of the R wave. Rate-dependent BBB.

Old anterior infarct. Long Q-T interval. Sinus bradycardia, 1° AV block, IVCD and prolonged Q-T interval. Short Q-T interval. Lateral MI. Acute inferoposterior MI, right-sided precordial leads revealing RV involvement. RVH, most likely from COPD. Atrial flutter. Increased vagal tone causing sinus node to slow down and AV block to occur. Mitral stenosis. Atrial fibrillation. MAT. Type II 2° AV block. Type I 2° AV block. Type I 2° AV block.

USEFUL TIPS FOR ECG INTERPRETATION

- *Quickly scan:*
 - Are the QRSs narrow or wide, occurring regularly or irregularly
 - P or no P
 - P-QRS relationship
- Some leads are more useful than others (e.g. pp. 44, 83)
- In a regular rhythm strip, if there is a break in regularity, start your analysis there, tackling one beat at a time.
- Sinus P wave is biphasic (initially positive then negative) with or without left atrial enlargement in V₁ and entirely upright in lead II.
- Among the 6 precordial leads, the most biphasic P wave belongs to V1. This fact will allow us to detect reversed precordial leads (e.g. pp. 129, 246, 252, 255)
- Before you call RAD (right axis deviation) check the P wave in lead I. If it is inverted, it is either reversed arm leads or dextrocardia. The precordial leads will settle the issue. (e.g. pp. 198, 252)
- If T wave is tented (pinched from above), consider hyperkalemia even if it is not tall. Sometimes only one lead is involved.
- The long QT in hypocalcemia is the result of T wave being pushed out from a long ST segment, which is different from other causes of long QT interval (e.g. p. 63)
- The short QT in hypercalcemia is the result of T wave being pulled in by a short ST segment (e.g. p. 64)
- *If a wide QRS is preceded by:*
 - No P wave, it is ventricular (e.g. pp. 140, 148, 190, 223, 235)
 - A regularly occurring P but with a shorter PR than others, it is ventricular (e.g. pp. 11, 41, 120, 289)
 - A premature P, it's PAC with aberrant conduction (e.g. pp. 67, 135)
- Group beatings; try to make Wenckebach phenomenon out of it (e.g. pp. 16, 42, 316)
- Findings in other leads can be helpful (e.g. pp. 61, 113)
- The most common unexpected pause is due to a nonconducted PAC (e.g. pp. 80, 279, 313, 351)
- Q wave in lead III; even if it is shallow, if it is wide, it is inferior MI. Even if it is deep, if it is skinny (narrow), ignore it. (e.g. pp. 82, 98)

SECTION 1



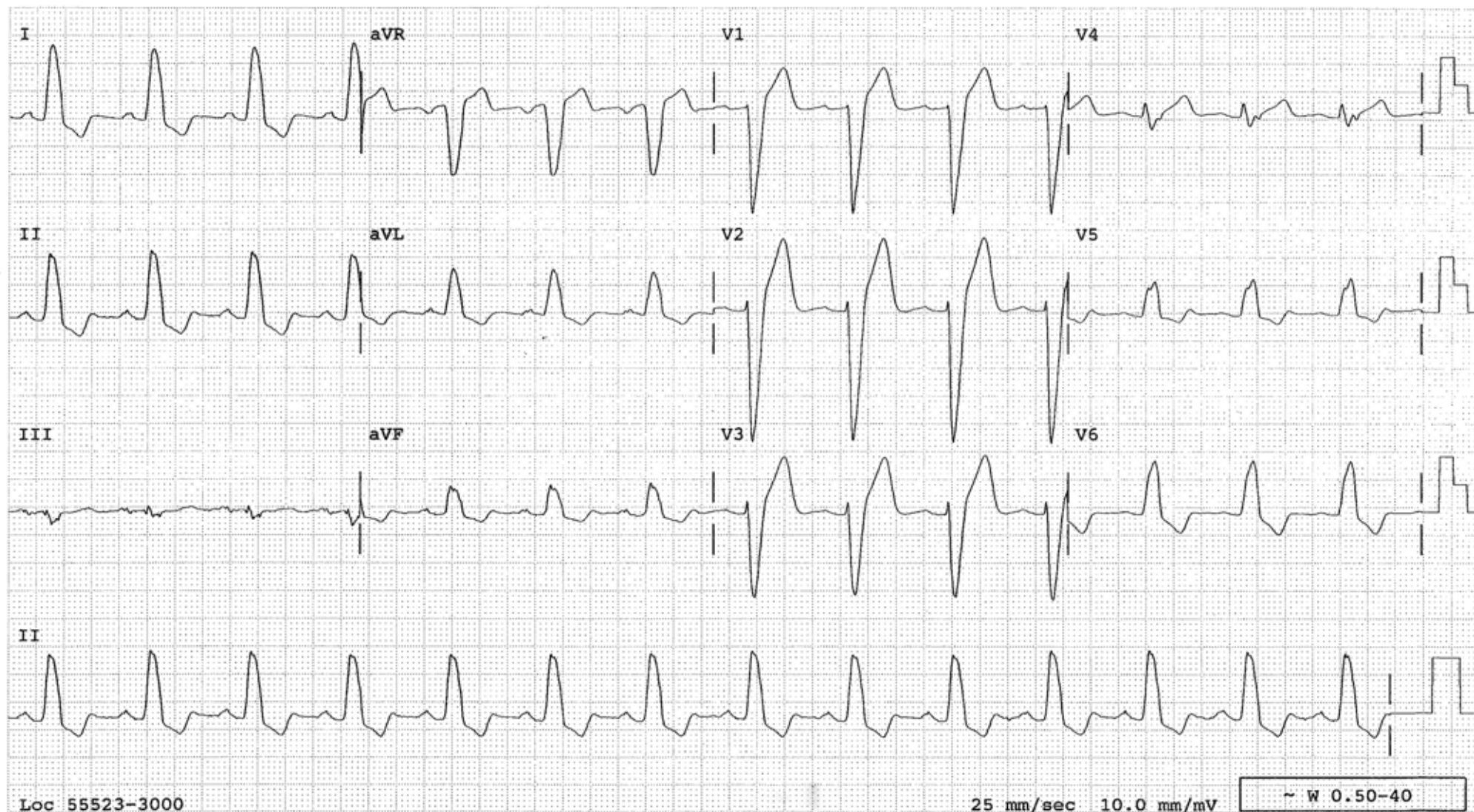
1-1 Let us analyze this tracing systematically. One should quickly see the calibration mark at the end and note that the tracing was taken with the standard calibration for both the limb (the first half of the mark) and precordial leads (the latter half of the mark). A regular rhythm at a rate of 65/minute is present. There is a positive P wave preceding each QRS with a fixed P-R interval, indicating normal sinus rhythm (NSR). The P-R interval of 0.16 seconds, the QRS duration of 0.1 seconds, the QRS voltage, the mean QRS axis and the Q-T interval of 0.4 seconds are all within normal range. The R waves (or R/S ratio) progress normally in the precordial leads and the transition (the change from R/S ratio of < 1 to > 1) occurring between V_3 and V_4 is normal, i.e. neither early nor late transition. Small Q waves in leads I, II, III, aVF and V_4 through V_6 are normal septal Q waves. For the Q wave to be abnormal in these leads, it must be wider than 0.04 seconds. The interventricular septum, which is the first part of the ventricular myocardium to be depolarized, is depolarized from the patient's left to right and often slightly cephalad, resulting in an initial negative deflection (Q wave) in these leads. In lead V_2 , however, there are no normal septal Q waves and any Q wave is abnormal and needs explanation. The T waves are upright in all leads except in aVR. There is a 1 mm ST elevation in leads V_1 through V_3 . Approximately 90% of young men have 1-3 mm concave ST-segment elevation in these leads normally.

Conclusion: Normal electrocardiogram (ECG)



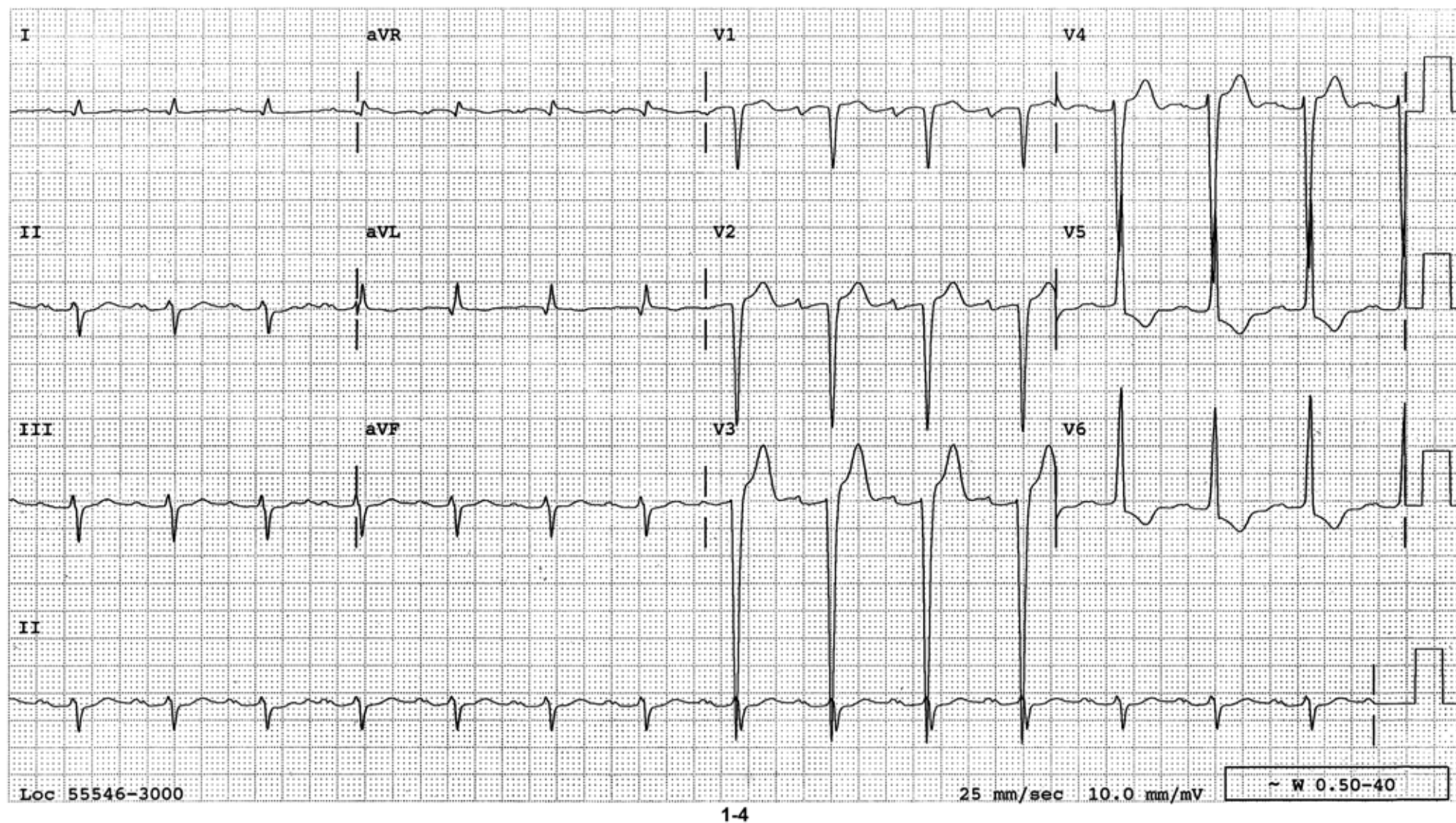
1-2 Normal sinus rhythm at a rate of 71/minute. The P wave is mostly negative in V_1 but upright in lead II, indicating left atrial enlargement (LAE). rsR' pattern in V_1 combined with broad S waves in leads I, aVL and V_6 , and the QRS duration of 130 milliseconds are all diagnostic features of complete right bundle branch block (RBBB). In RBBB, the right ventricle is depolarized delayed resulting in broad S waves in these leads and the R' in V_1 .

- Dx:
1. NSR
 2. RBBB
 3. LAE



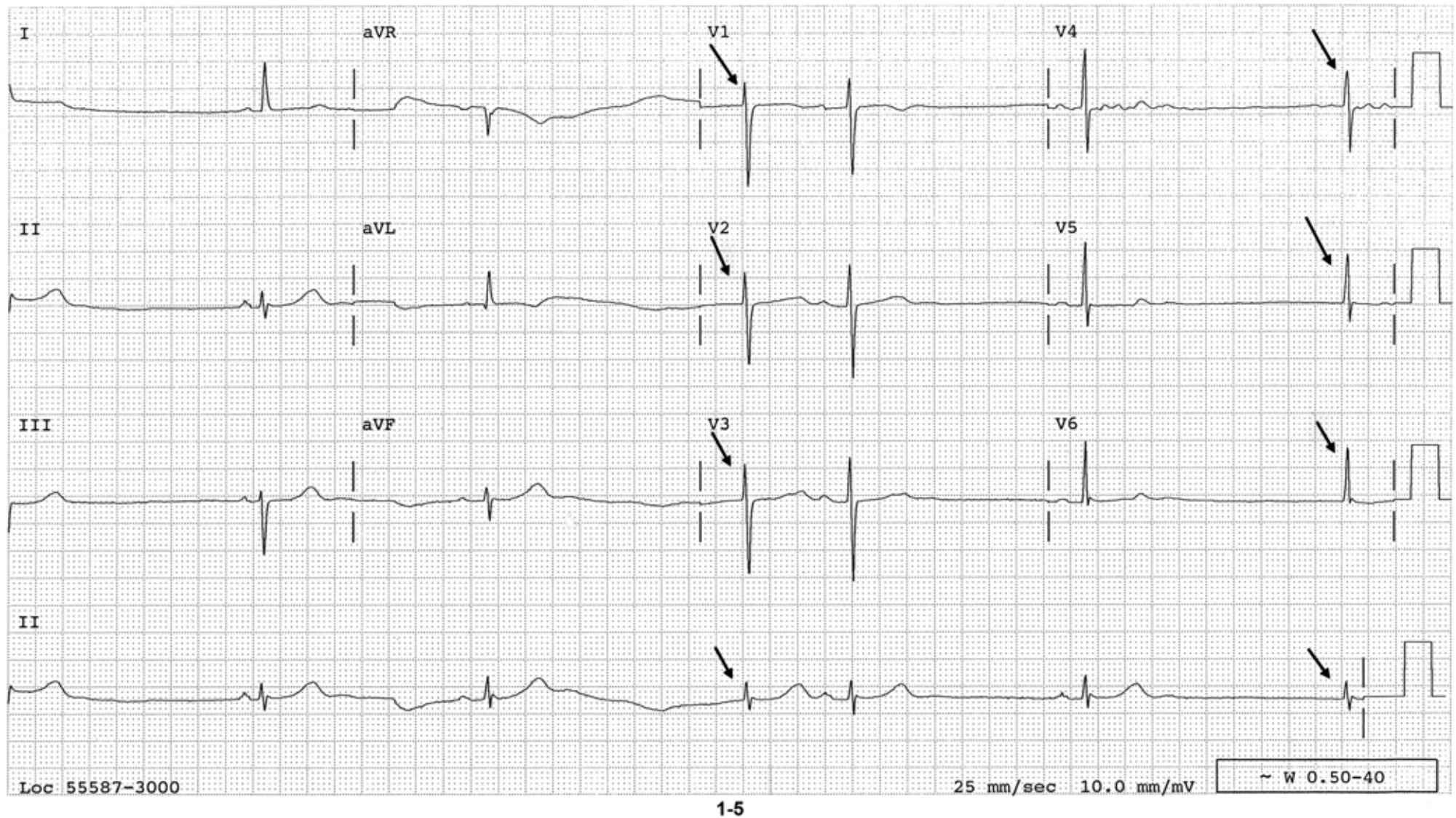
- 1-3 Normal sinus rhythm at a rate of 84/minute. The QRS is wide and measures 148 milliseconds. Monophasic R waves in leads I, aVL and V₆ combined with mostly negative QRSs in the right precordial leads are diagnostic of left bundle branch block (LBBB). In LBBB, the ventricles are depolarized from the patient's right to left from the beginning to the end, which is responsible for these ECG manifestations. Marked ST elevation or ST depression seen in this tracing is all secondary to LBBB. Note that these secondary ST-T changes are directed opposite to the main QRS component in every lead. If the ST-T changes are directed to the same direction as the main QRS component (concordant ST-T changes), it will be called primary ST-T changes, which may be due to myocardial ischemia or infarction.

Dx: 1. NSR
2. LBBB



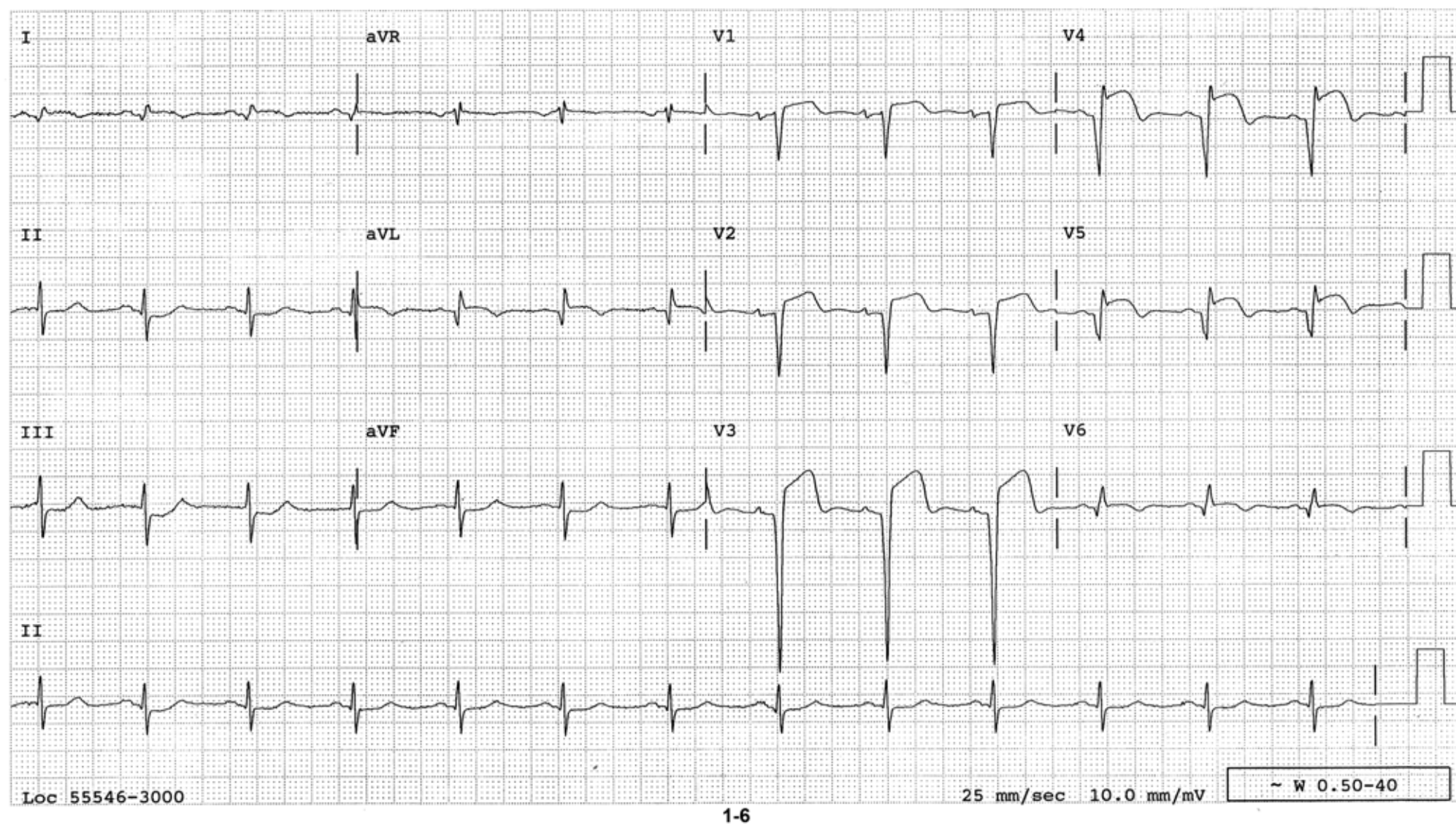
1-4 Normal sinus rhythm at 87/minute. The P-R interval is prolonged to 246 milliseconds indicating 1° atrioventricular (AV) block. The P wave is mostly negative in V₁ indicating LAE. The QRS axis is shifted to the left indicating left anterior fascicular block (LAFB) [left axis deviation is not part of left ventricular hypertrophy (LVH)]. Voltage criteria and ST-T changes for LVH are present. QS pattern in V₁ and V₂ and only a tiny R wave in V₃ may be secondary to LVH with or without old anteroseptal myocardial infarction (AMI). The ST-segment is elevated in V₃. This degree of concave ST-segment elevation can be normally seen especially when the S wave is deep.

- Dx:
1. NSR
 2. 1° AV block
 3. LAE
 4. LVH
 5. LAFB



1-5 The sinus rhythm is very slow and irregular with occasional long pauses allowing the AV junctional pacemaker to escape (third and sixth complexes). The sinus node is not functioning properly, hence sinus node dysfunction. This may be secondary to medications, or may be primary. If it is primary, it is called sick sinus syndrome.

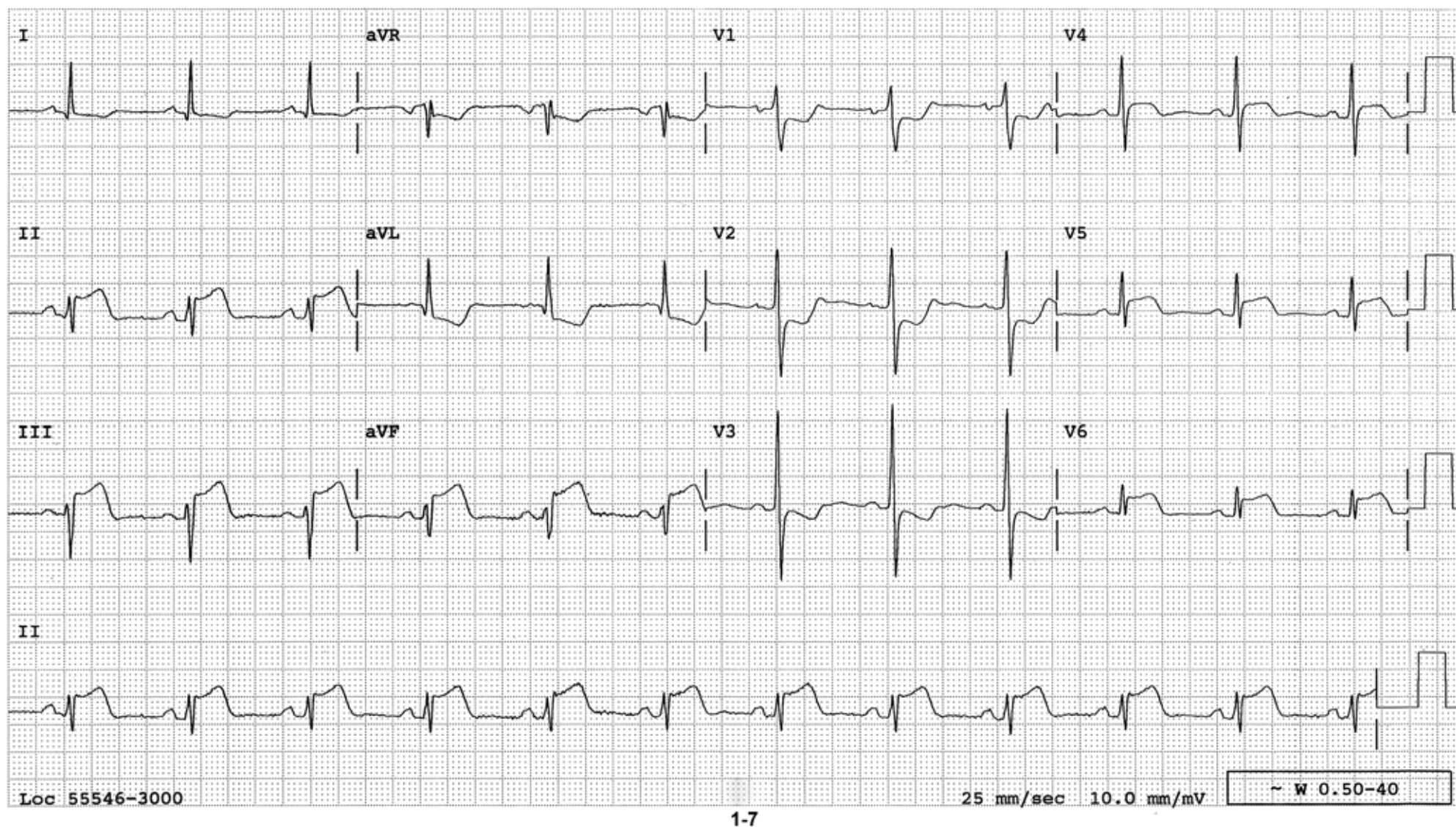
Dx: Sinus node dysfunction with occasional AV junctional escape complexes



1-6 Normal sinus rhythm at a rate of 78/minute. Q waves with ST elevation and terminally inverted T waves in the precordial leads as well as in leads I and aVL reflect extensive acute anterior myocardial infarction (MI). Mild ST depression in lead III is the reciprocal change of the ST elevation in aVL.

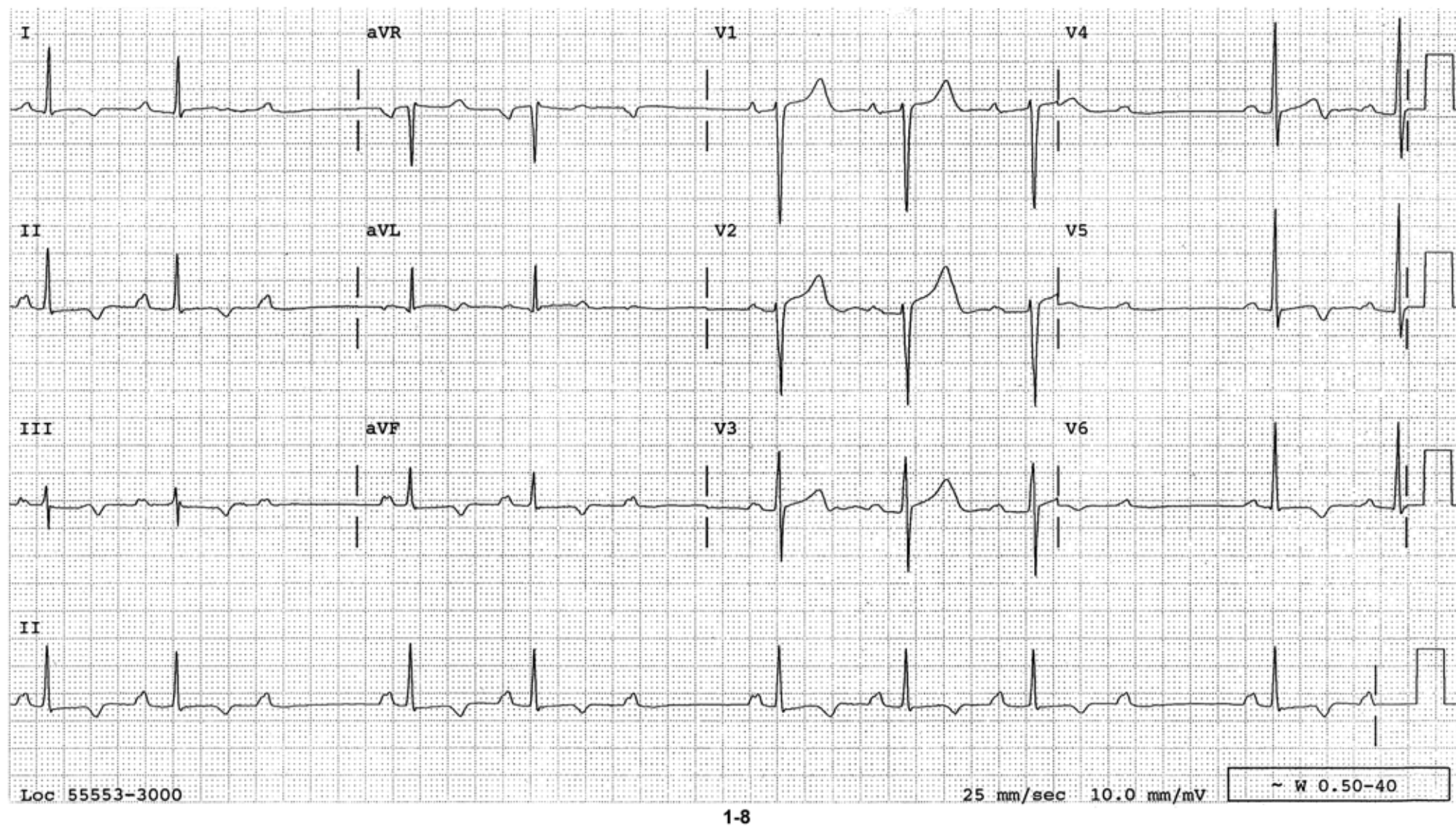
Dx: 1. NSR

2. Acute, extensive anterior infarct



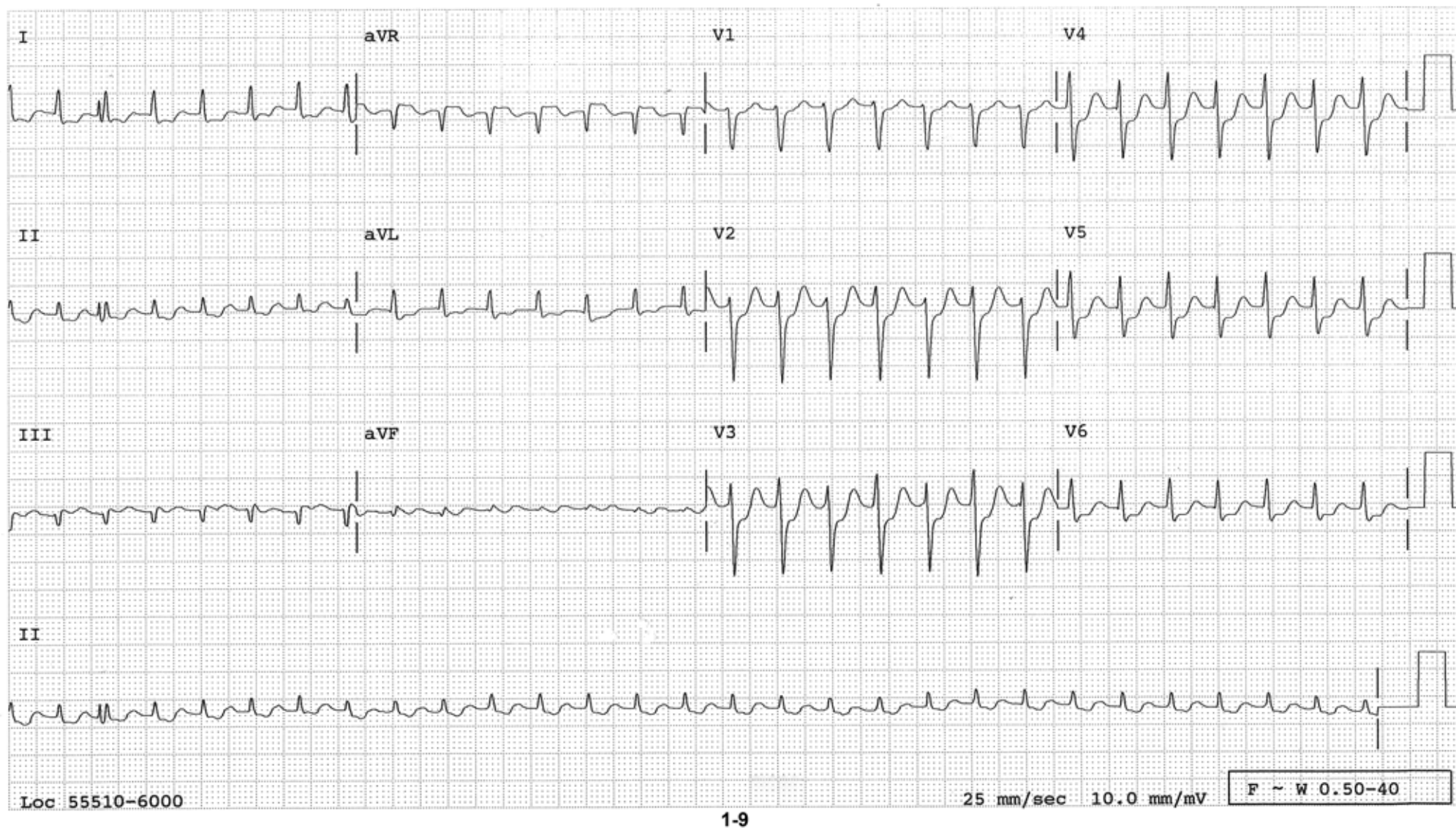
1-7 Normal sinus rhythm at a rate of 72/minute. ST-segment is elevated in the inferolateral leads reflecting acute inferolateral infarction. The ST-segment is horizontally depressed in the right precordial leads. This, combined with tall R waves in these leads, indicates reciprocal change of posterior wall involvement. Thus, this patient has an acute infarction involving the electrocardiographic area of inferoposterolateral wall.

- Dx:*
1. NSR
 2. Acute inferoposterolateral infarct



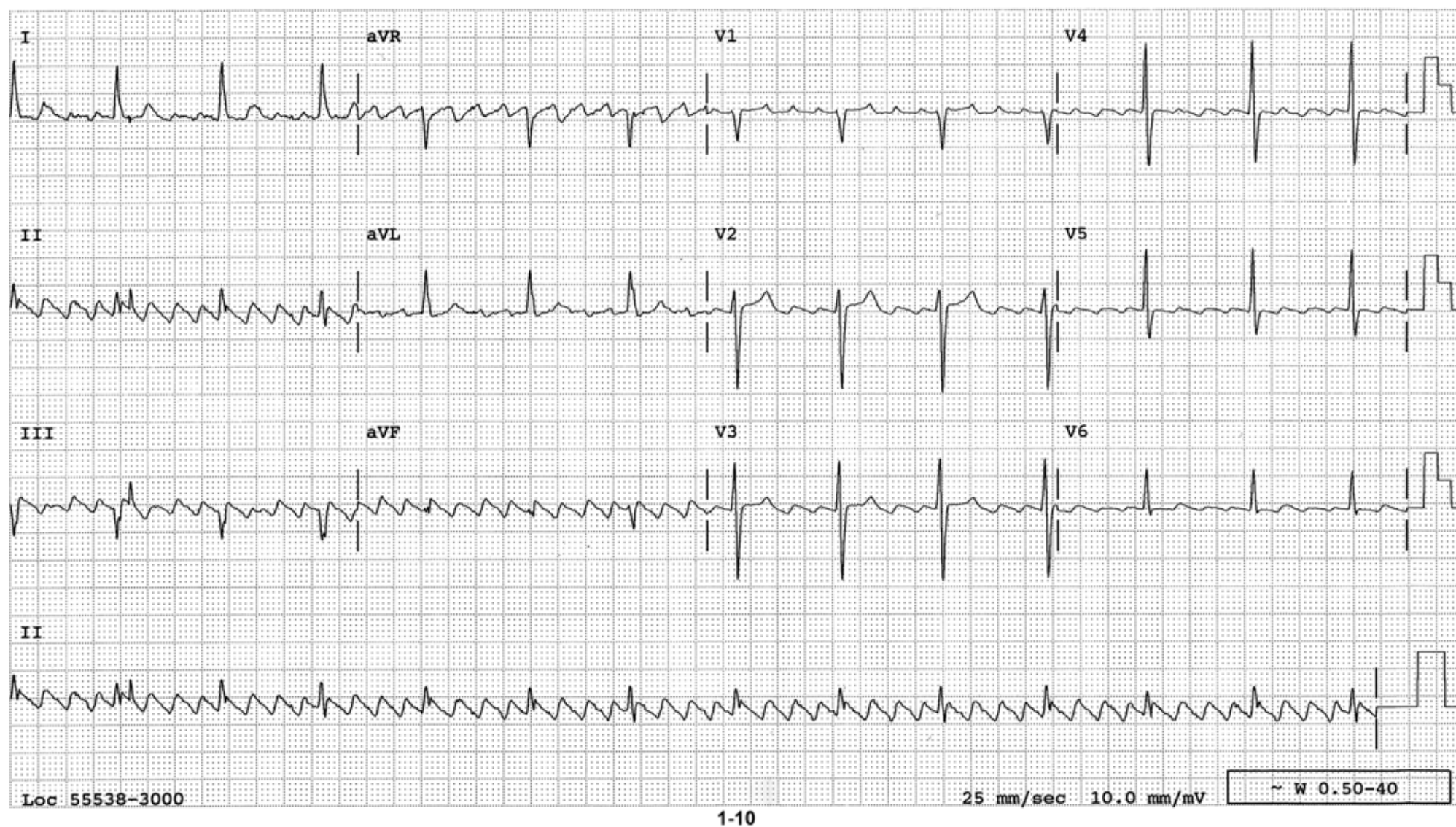
1-8 The atria are under the control of sinus node at a rate of 65/minute. At times, the P wave is blocked. The P-R interval progressively lengthens before the P wave is blocked and this is a typical example of Type I 2° AV block (AV Wenckebach phenomenon). The ST-segment is slightly elevated in V_2 which can be normally seen. T waves are inverted in the inferolateral leads suggesting either myocardial ischemia or even subendocardial infarction.

- Dx:*
1. Type I 2° AV block
 2. T wave changes



1-9 A narrow QRS tachycardia at a rate of 172/minute. No P waves are clearly discernible and the rhythm can be called supraventricular tachycardia (SVT) in a broad sense which includes ectopic atrial tachycardia, junctional tachycardia, AV junctional reentrant tachycardia (~ 60% of SVTs) or AV reentrant tachycardia using accessory pathway (~ 30% of SVTs). Horizontal ST depression in the mid-precordial leads may be secondary to tachycardia alone, or myocardial ischemia induced by the tachycardia. The Q wave in lead III is deep and wide enough to raise the possibility of an old inferior MI. The QRS complexes alternate especially in V_3 [electrical alternans (EA)]. EA is sometimes seen during SVT, atrial flutter or ventricular tachycardia (VT) in the absence of pericardial problems. Only during sinus rhythm, EA indicates cardiac tamponade.

- Dx:
1. SVT
 2. Consider an old inferior infarct
 3. EA
 4. ST depression, which may or may not be due to ischemia



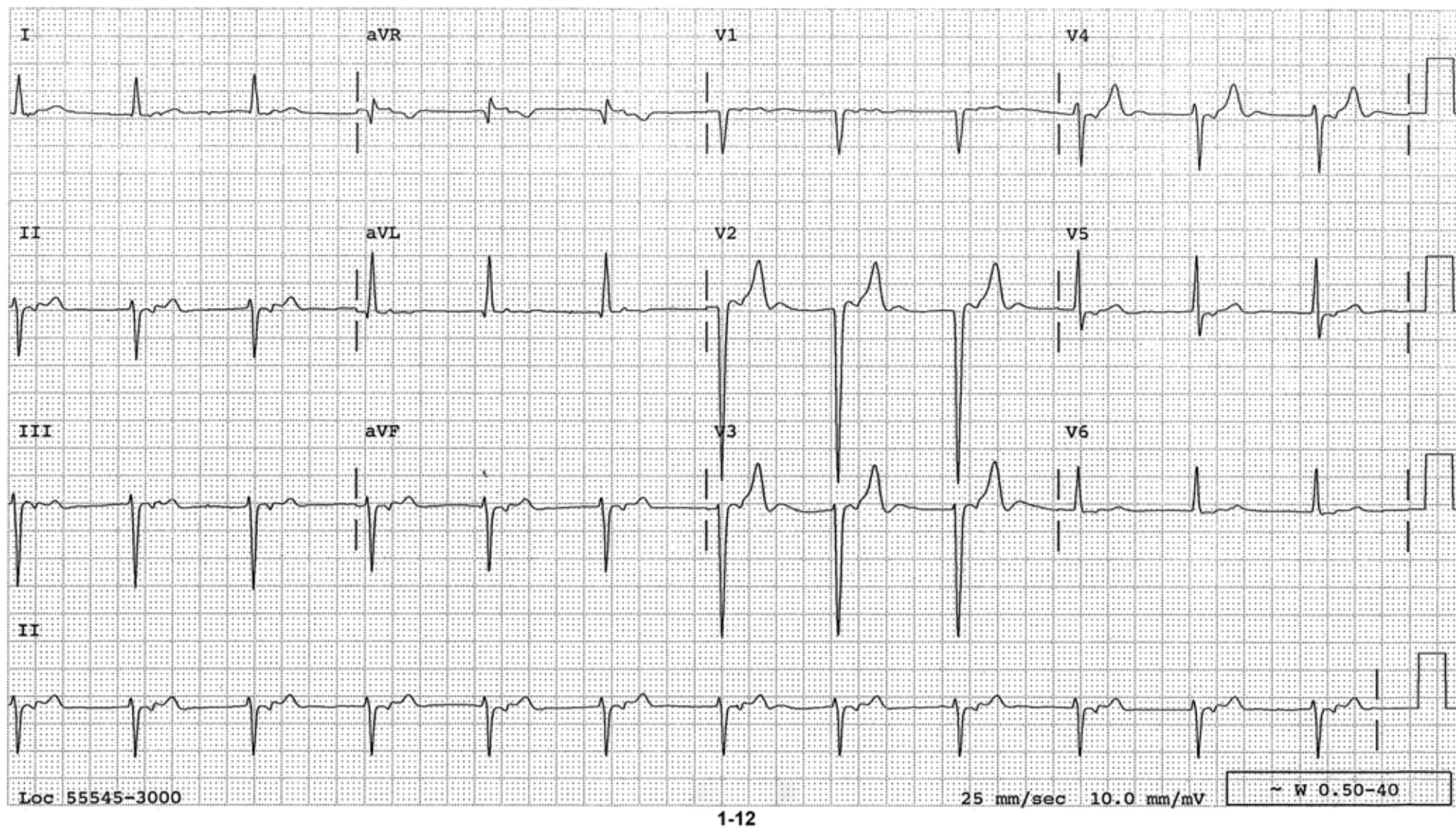
1-10 Atrial flutter with 4:1 AV conduction. As a rule, the sawtooth pattern of atrial flutter is primarily seen in the inferior leads as in this case. The precordial leads are half-standard and has voltage criteria for LVH. The Q wave in lead III raises the possibility of old inferior MI.

- Dx:*
1. Atrial flutter with 4:1 AV conduction
 2. LVH
 3. Consider old inferior infarct



1-11 This rhythm strip reveals a regular sinus rhythm at a rate of about 90/minute. The P-R interval is prolonged to 220 millisecond making 1° AV block. Three wide QRSs are present. Even though they are preceded by a P wave, the P-R interval is shorter than other conducted sinus complexes indicating that these QRSs are not induced by the P wave. They are late occurring ventricular premature complexes. They are not premature atrial contractions (PACs) with aberrant conduction because the P waves do not occur prematurely and the P-R interval is too short for the P wave to have conducted to the QRS.

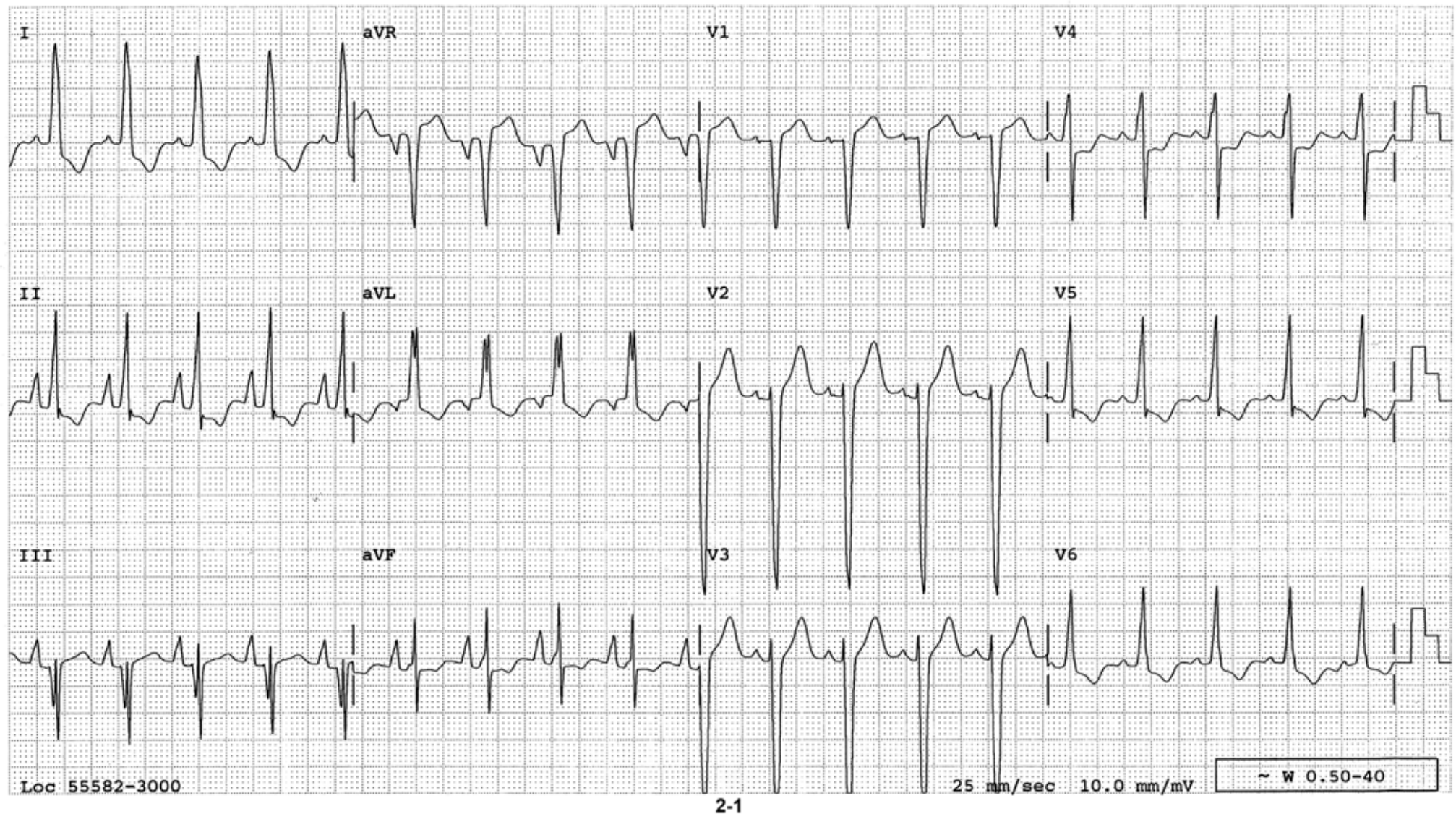
- Dx:*
1. NSR with 1° AV block
 2. Premature ventricular contractions (PVCs)



1-12 A regular rhythm at a rate of 70/minute. The QRSs are narrow indicating that they originated from above the bifurcation of the intraventricular conduction system. There is no P wave in front of the QRS. However, there is a negative deflection immediately following the QRS, especially noticeable in the inferior leads indicating retrograde activation of the atria. This is a good example of AV junctional rhythm with 1:1 conduction to the atria. Since the rate is not faster than 130/minute, it is not junctional tachycardia. However, since the rate is faster than the intrinsic AV junctional rate of 40–50/minute, this rhythm will be called accelerated junctional rhythm. Accelerated junctional rhythm occurs with digitalis intoxication, myocardial ischemia or infarction or excess amount of catecholamines circulating, which means any stressful condition. The mean QRS axis is shifted to the left reflecting LAFB. The QRS voltage is increased suggesting LVH. Q waves in V_1 and V_2 may be due to LVH with or without old AMI.

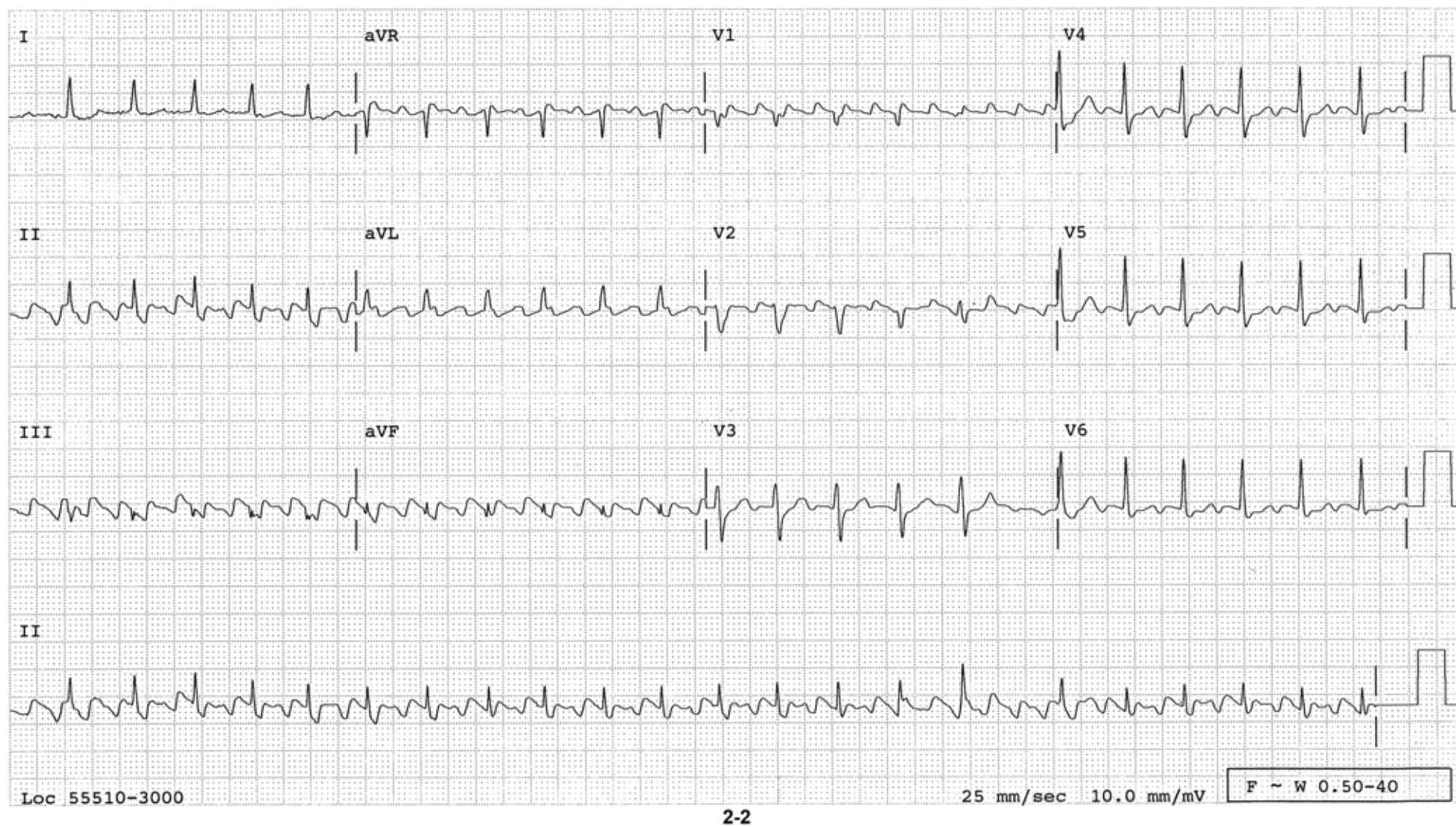
- Dx:
1. Accelerated AV junctional rhythm with 1:1 retrograde conduction to the atria
 2. LAFB
 3. LVH by voltage
 4. Poor R wave progression 2° to LVH with or without old anteroseptal infarct

SECTION 2



2-1 Sinus tachycardia at a rate of 113/minute. The P wave is very tall in lead II (6 mm) reflecting right atrial enlargement (RAE). Voltage criteria and ST changes for LVH are present. Note that the precordial leads are half-standard. It is not unusual for the ST-segment to be elevated in V_2 normally, especially when it is accompanied by a deep S wave. The upstroke of the QRS in aVF is slurred but, judging from aVR and aVL taken simultaneously, that is the beginning of a normally conducted QRS rather than a delta wave. The Q wave in lead III is deep and wide enough to make one consider old inferior MI.

- Dx:
1. Sinus tachycardia
 2. RAE
 3. LVH
 4. Consider old inferior infarct



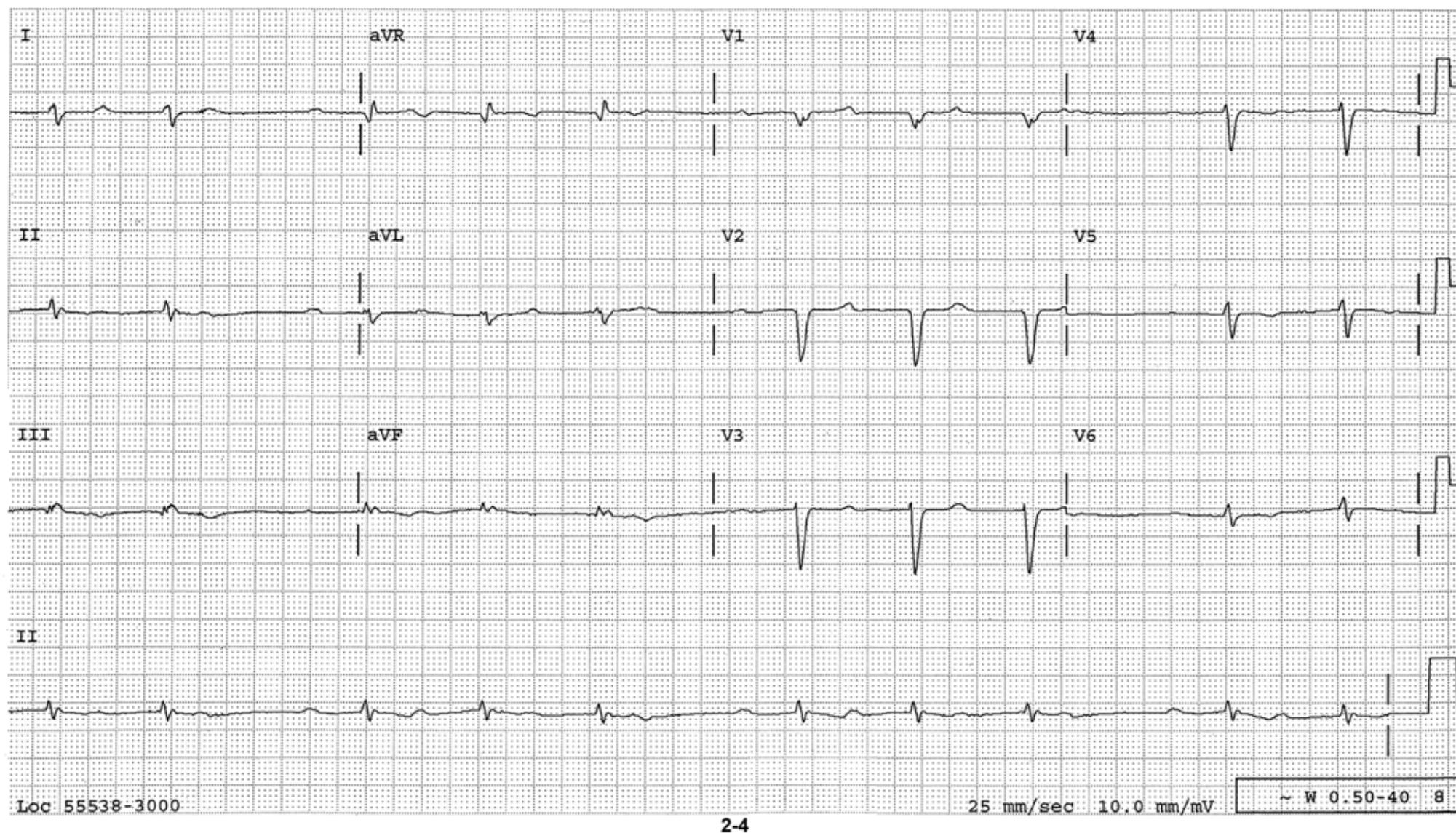
2-2 A narrow QRS tachycardia at a rate of 138/minute. The tracing is highly suggestive of atrial flutter with 2:1 AV conduction. This is proven by the fact that, when there is 4:1 AV conduction toward the end of the tracing, intact flutter waves are readily revealed.

Dx: Atrial flutter with mostly 2:1 and occasional 4:1 AV conduction



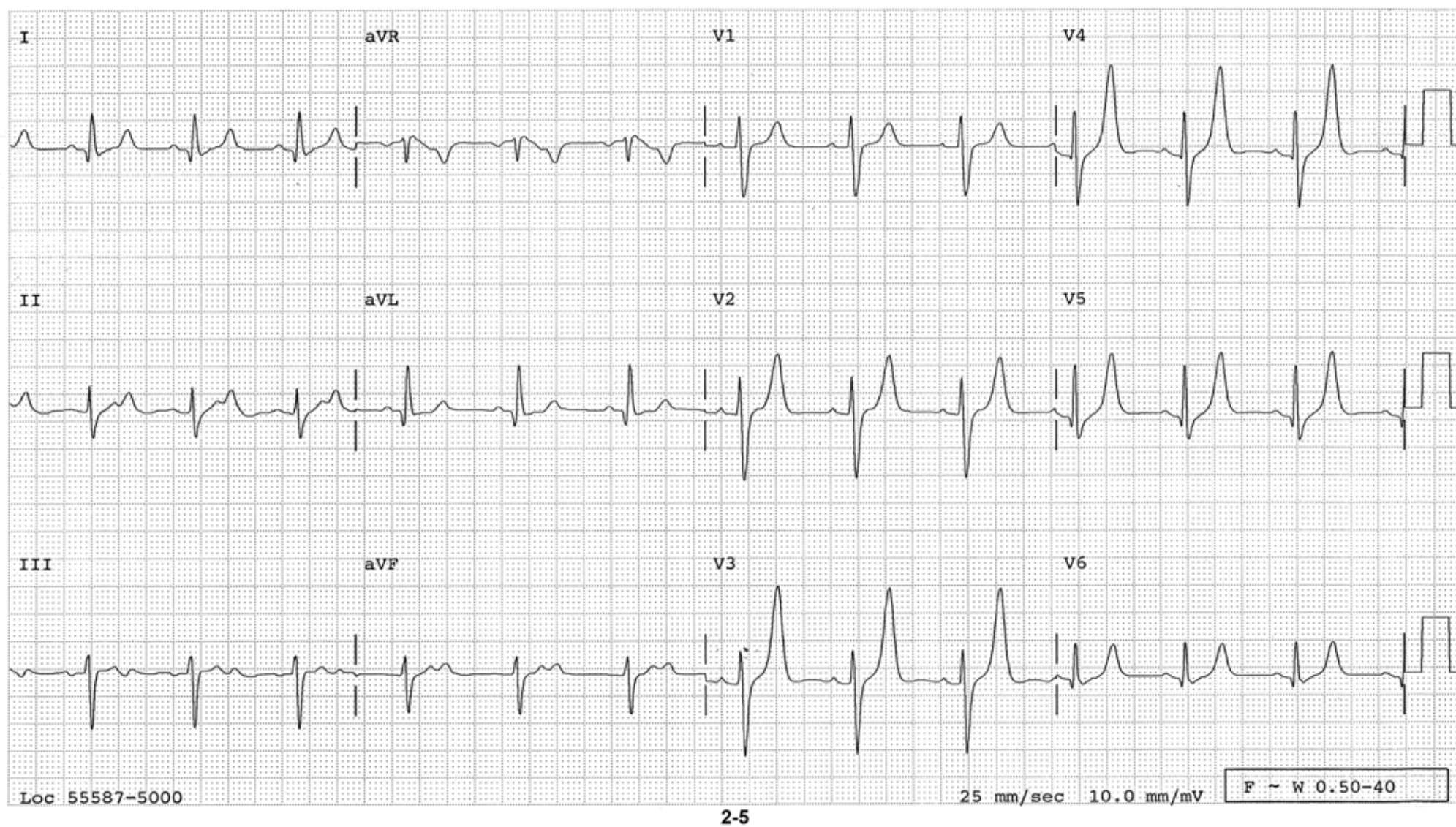
2-3 The first part of the tracing raises the possibility of atrial flutter, but lead III taken simultaneously reveals sinus rhythm convincingly. Muscle tremor or other artifacts can simulate atrial flutter as in this patient. The R waves progress poorly in the right precordial leads raising the possibility of old AMI.

- Dx:*
1. NSR
 2. Artifact simulating atrial flutter
 3. Consider old anteroseptal infarct



2-4 There is a group beating of the QRSs, which should always raise the possibility of Wenckebach phenomenon. Indeed the P-R interval progressively lengthens until the fourth P wave is blocked. This is best appreciated in the rhythm strip of lead II. The QRS axis is shifted to the right. QRS voltage is low in the limb leads (no QRS is taller than 5 mm). There is a late transition in the precordial leads (the transition from R/S ratio < 1 to > 1 occurring after V_3 or V_4). These changes may be secondary to diffuse myocardial involvement with amyloidosis, fibrosis, or other conditions. The QRS measures 123 milliseconds but the QRS pattern does not fit either the right or LBBB and this will be called nonspecific intraventricular conduction defect (IVCD).

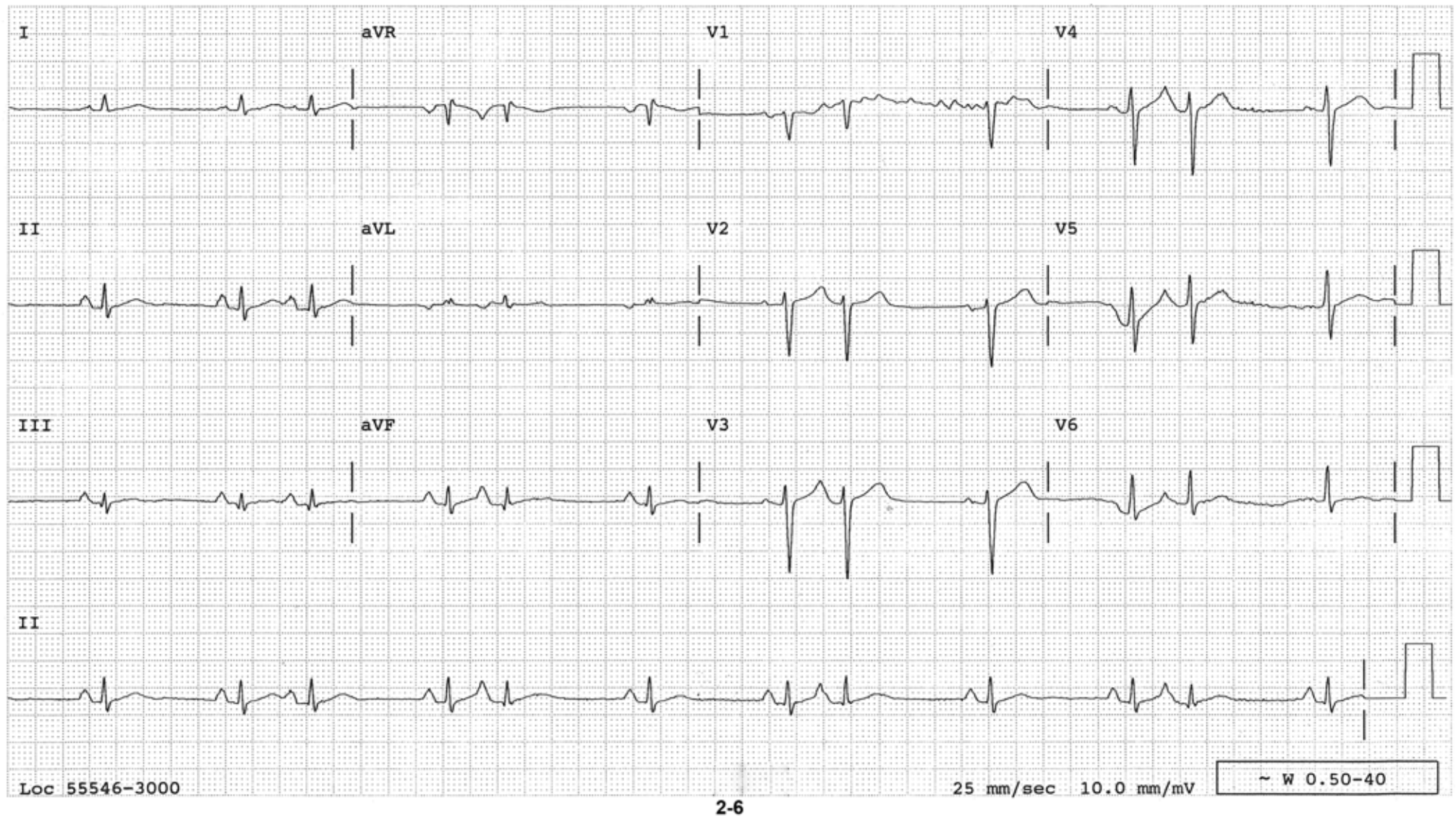
- Dx:
1. NSR with Type I 2° AV block
 2. Right axis deviation (RAD)
 3. Low QRS voltage
 4. Late transition
 5. IVCD



2-5 Sinus rhythm at a rate of 75/minute. The T waves are very tall, narrow, pointed and tented. This tracing is diagnostic of hyperkalemia.

Dx: 1. NSR

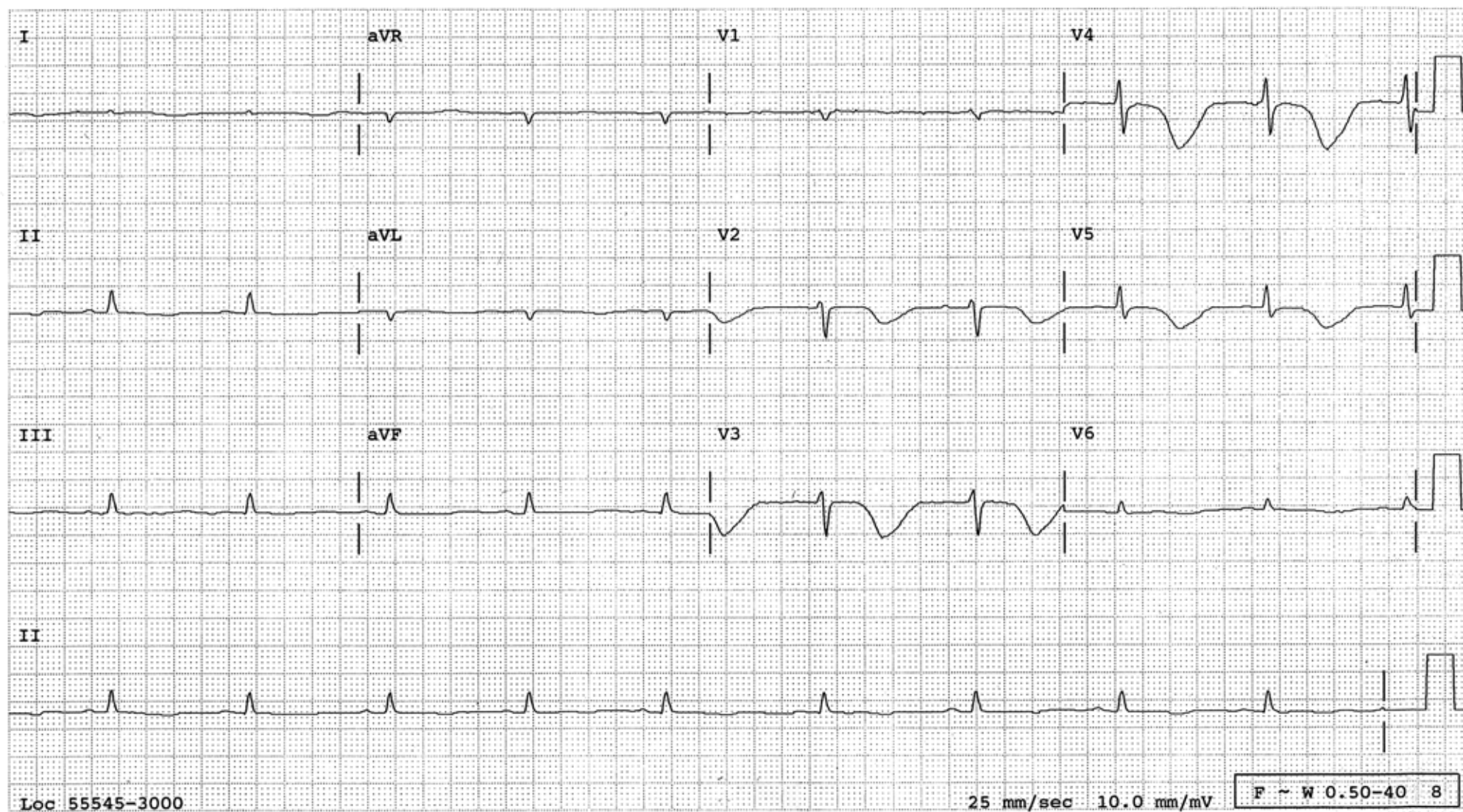
2. Hyperkalemia



2-6 Sinus rhythm with frequent premature complexes. These premature complexes are preceded by a P wave which many times is superimposed on the T wave.

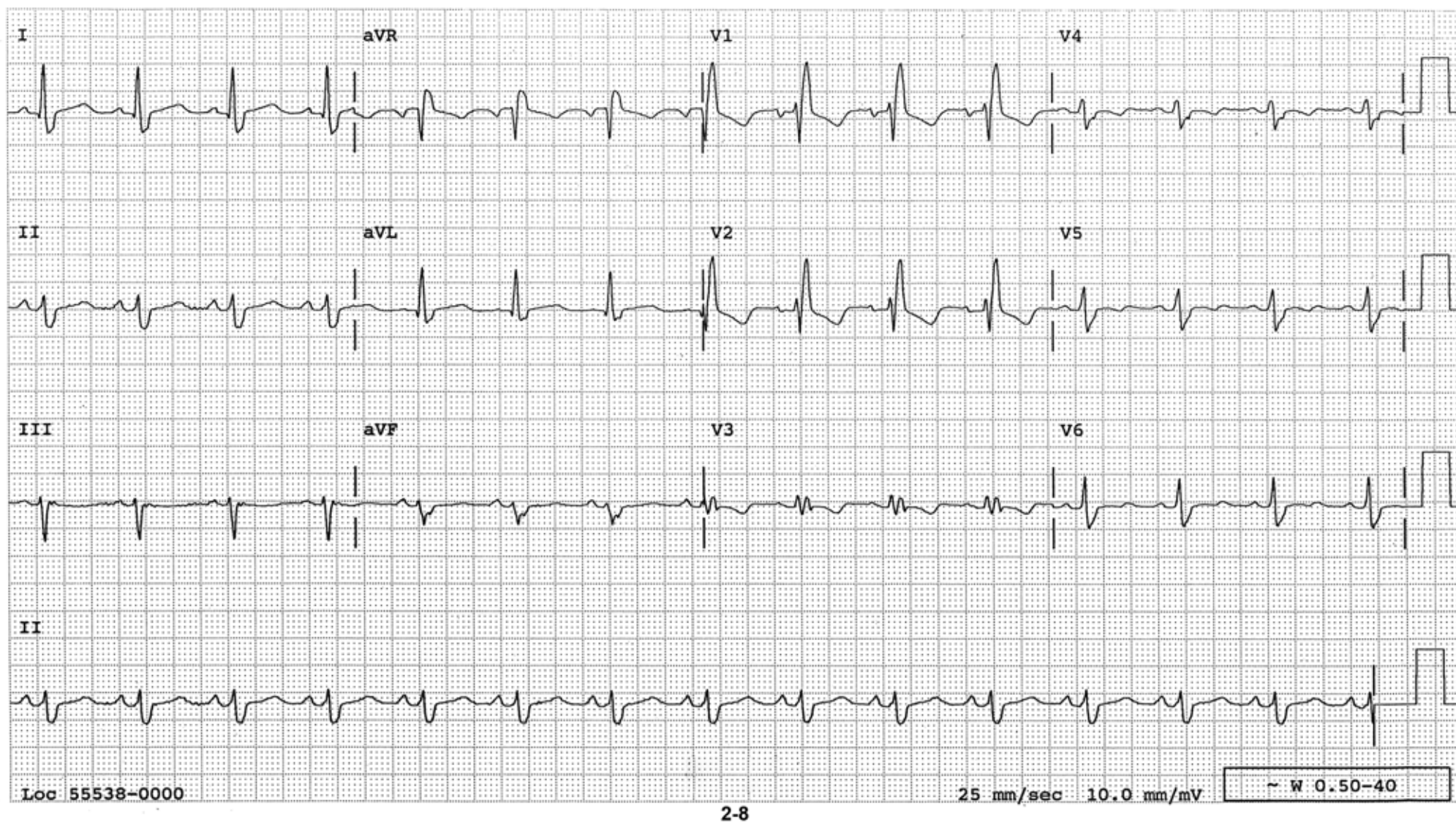
Dx: 1. NSR

2. Multiple PACs



2-7 Normal sinus rhythm at a rate of 57/minute. There is a low voltage in the limb leads. The striking abnormalities are the T wave inversion and a very long Q-T interval measuring about 760 milliseconds. This may be secondary to medications or stunned myocardium from a variety of conditions, such as central nervous system (CNS) events or any other causes of catecholamine surge (stress cardiomyopathy). This patient is at risk of developing *Torsade de Pointes*.

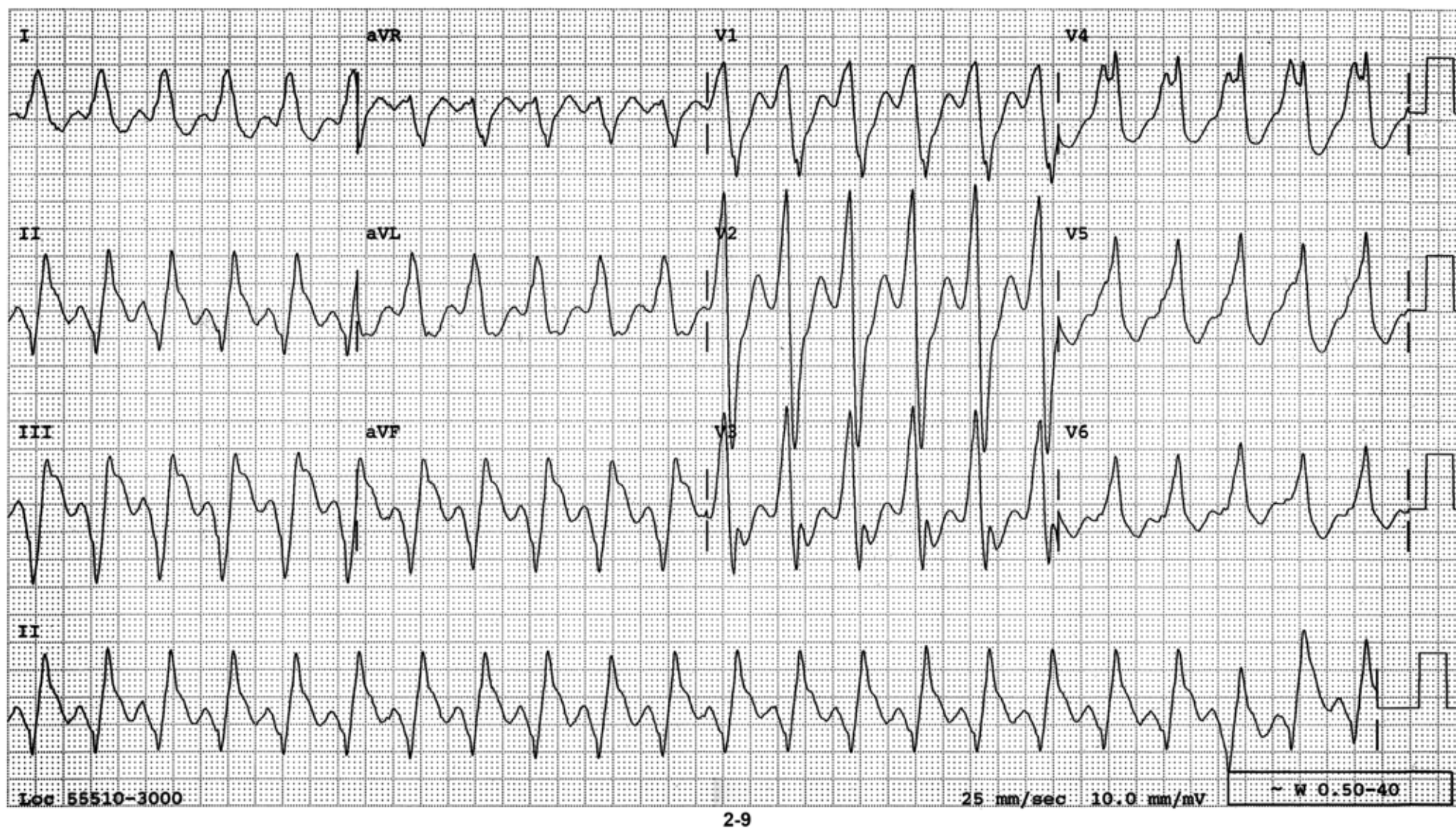
- Dx:
1. NSR
 2. Low QRS voltage
 3. T wave inversion
 4. Long Q-T interval
 5. Consider stress cardiomyopathy



2-8 Sinus rhythm at a rate of 85/minute. Wide QRS complexes, rsR' in V_1 - V_2 and broad S waves in leads I, aVL and V_6 are diagnostic of RBBB. The mean QRS axis is shifted to the left, indicating LAFB; hence, bifascicular block (BIFB).

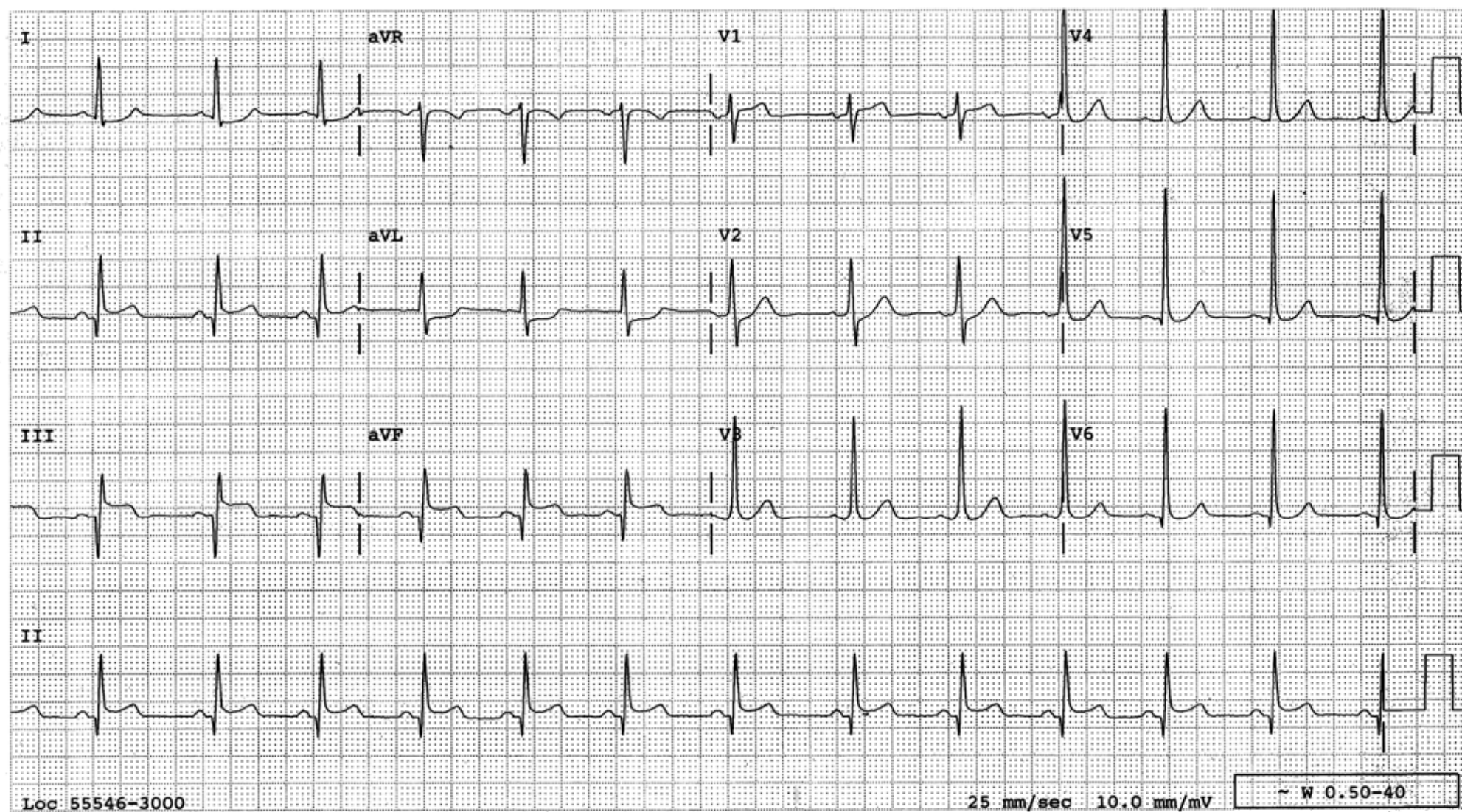
Dx: 1. NSR

2. Bifascicular block consisting of complete RBBB and LAFB



2-9 Wide QRS tachycardia at a rate of 131/minute. Wide QRS tachycardia is either SVT with aberrant conduction or SVT with pre-existing bundle branch block (BBB) or VT. The P waves are not clearly identified and one cannot establish either AV dissociation or association. The QRS is markedly widened to 240 milliseconds and the tracing is strongly suggestive of VT. In aberrant conduction, the QRS width seldom exceeds 140 milliseconds. The QR pattern with ST elevation in inferior leads is so characteristic of infarction pattern that either acute inferior MI or, if it is a chronic finding, a ventricular aneurysm can be called even from the QRSs of VT. Same thing can be said from the QRSs of accelerated idioventricular rhythm, ventricular paced rhythm and PVCs. This patient proved to have a ventricular aneurysm.

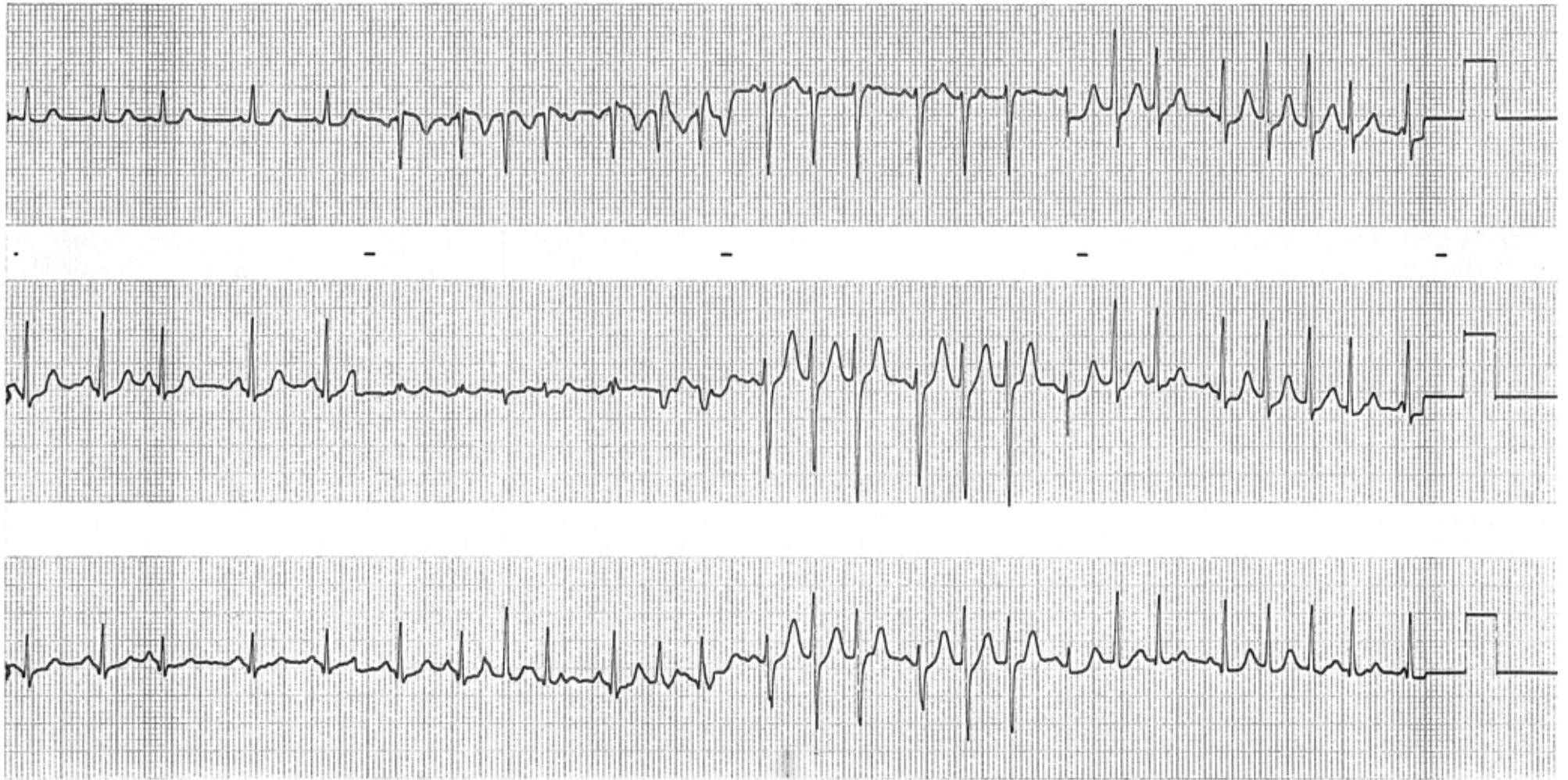
- Dx: 1. VT
2. Either acute inferior ST-segment elevation myocardial infarction (STEMI) or, if the findings are old, ventricular aneurysm.



2-10

2-10 NSR at a rate of 80/minute. Deep Q waves with ST elevation in the inferior leads combined with tall R waves in the right precordial leads indicate inferoposterior MI. ST-segment is slightly elevated in V_1 raising the possibility of right ventricular (RV) infarction as well. This can be verified by obtaining right-sided precordial leads.

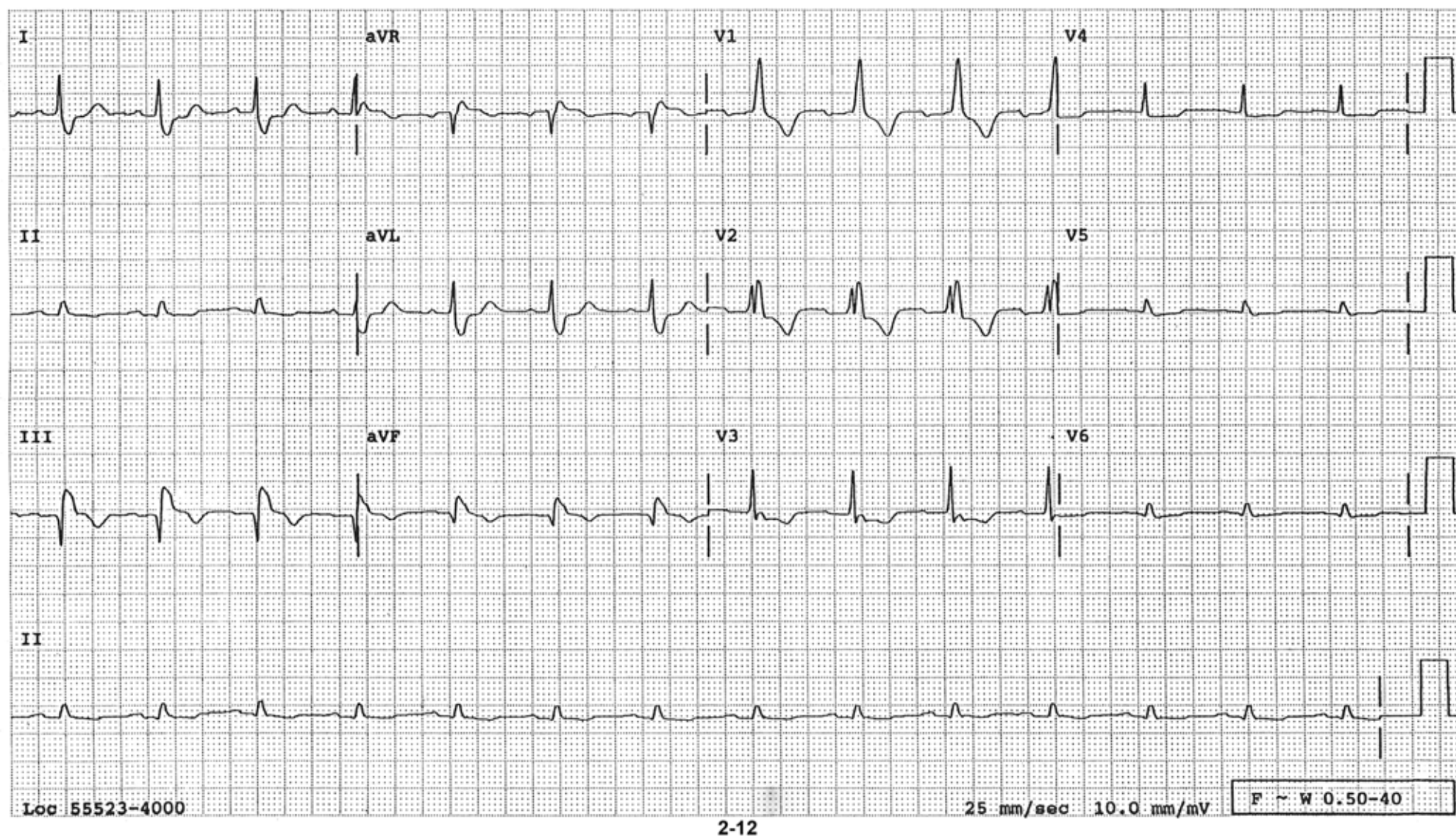
- Dx:
1. NSR
 2. Acute inferoposterior infarct
 3. Consider RV infarct



2-11

2-11 Irregularly irregular rhythm at a rate of 160/minute. The QRSs are narrow and are preceded by a P wave of changing morphology. The P waves occur irregularly. These are diagnostic features of multifocal atrial tachycardia (MAT).

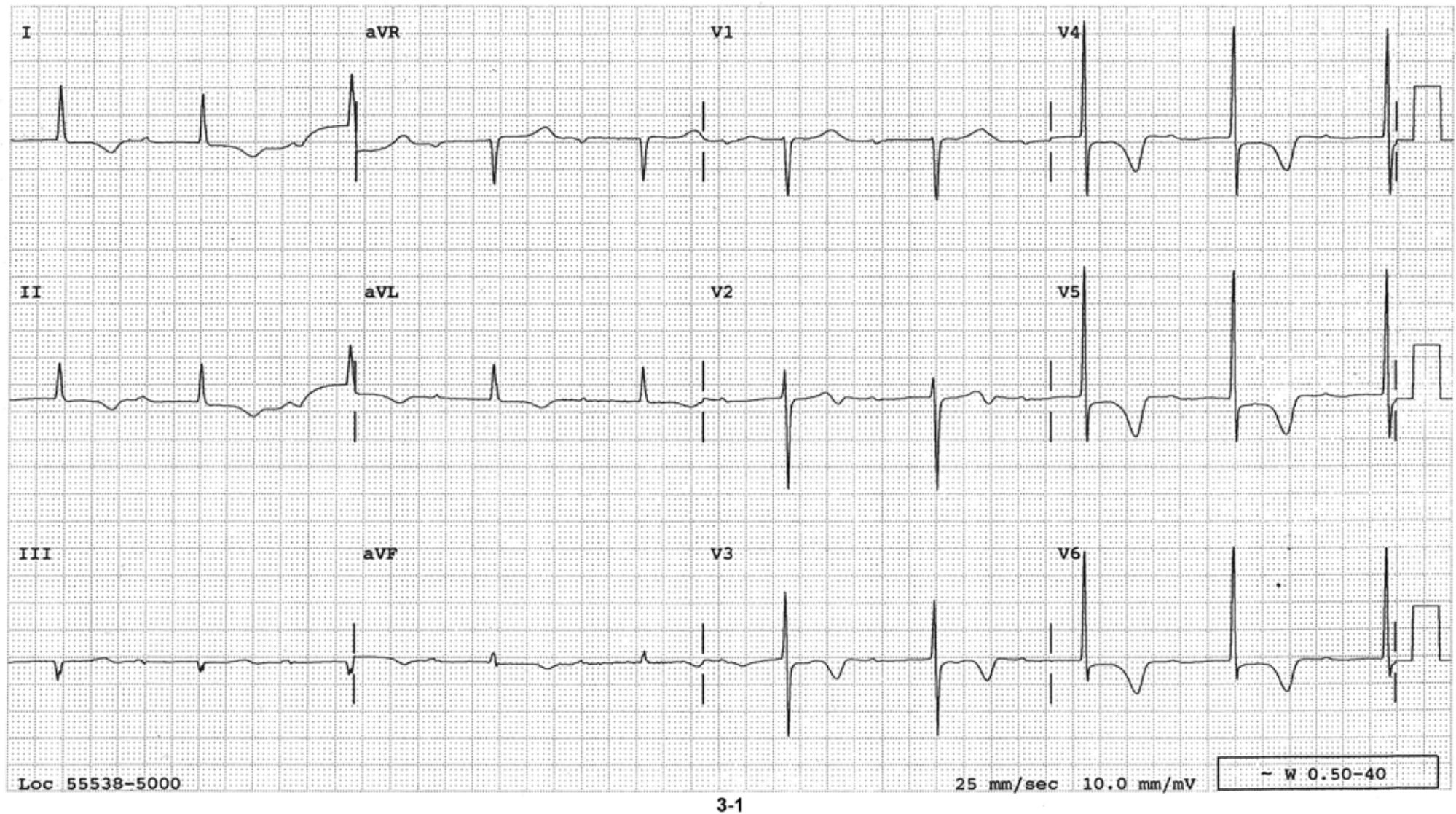
Dx: MAT



2-12 Sinus rhythm at a rate of 80/minute. RBBB is present. Q waves in the inferior leads with ST elevation and T wave inversion indicate acute inferior MI. RBBB affects only the latter portion of the QRS while infarction affects only the initial portion of the QRS. Therefore, RBBB does not interfere with the manifestation of Q wave infarction.

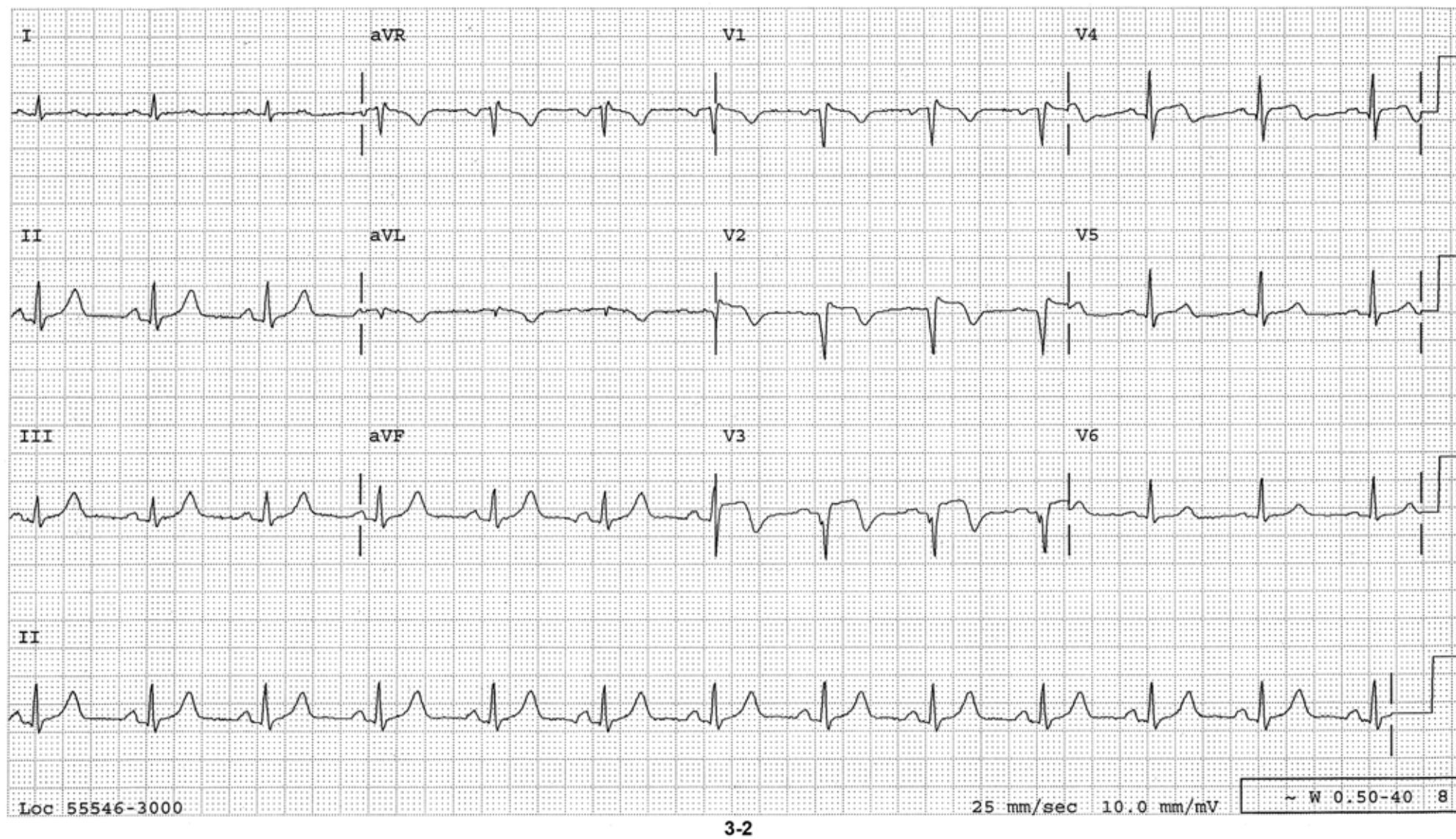
- Dx:
1. NSR
 2. RBBB
 3. Acute inferior infarct

SECTION 3



3-1 Normal sinus rhythm at a rate of 56/minute. The P-R interval is prolonged to 460 milliseconds, but every P wave results in a QRS making 1° AV block. Voltage criteria and ST-T changes for LVH are present. The T waves are inverted more than usual for LVH and one has to consider myocardial ischemia additionally.

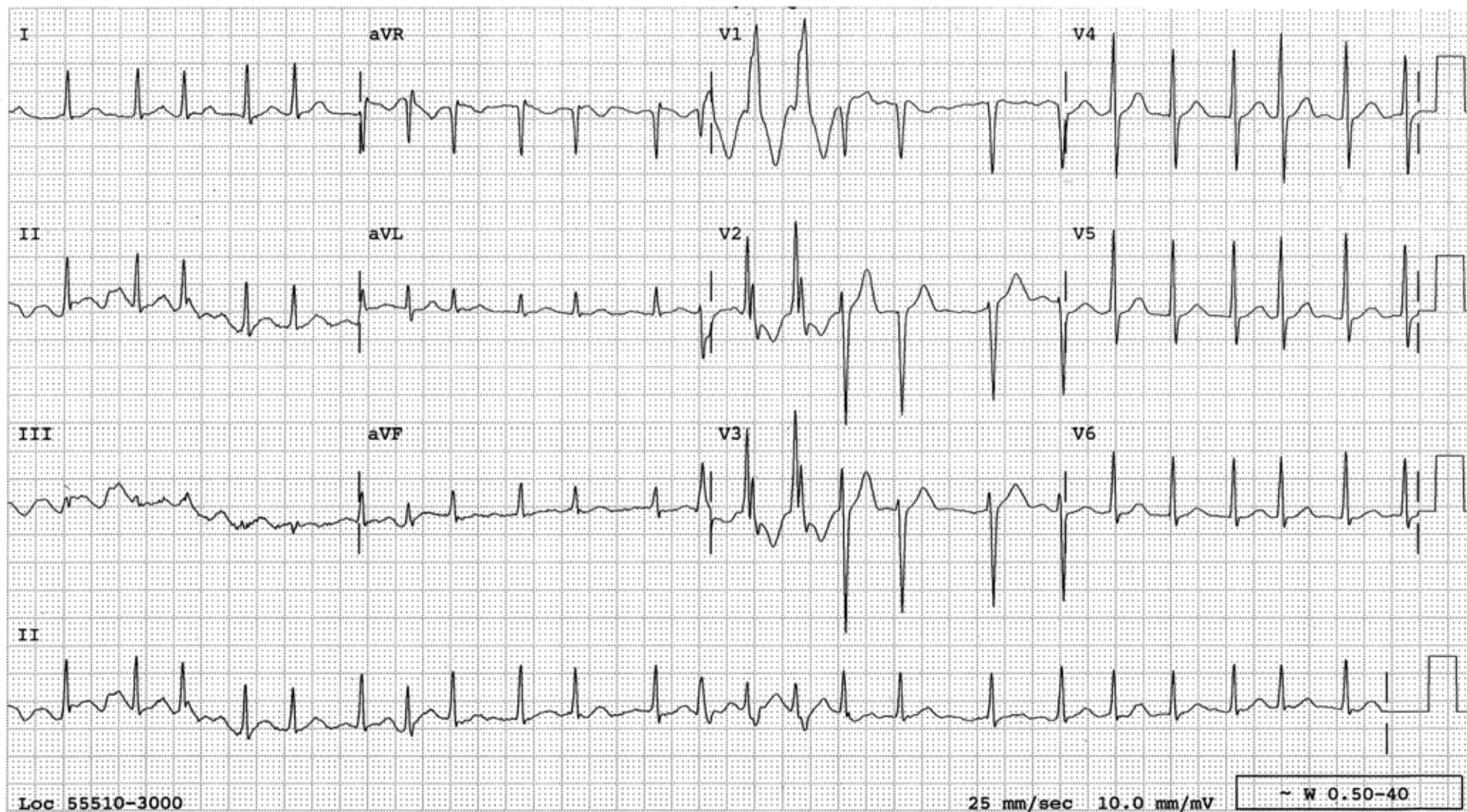
- Dx:
1. NSR
 2. 1° AV block
 3. LVH
 4. T wave inversion suggestive of ischemia



3-2 Normal sinus rhythm at a rate of 75/minute. Q waves with ST elevation and terminal T wave inversion in the right precordial leads reflect recent AMI.

Dx: 1. NSR

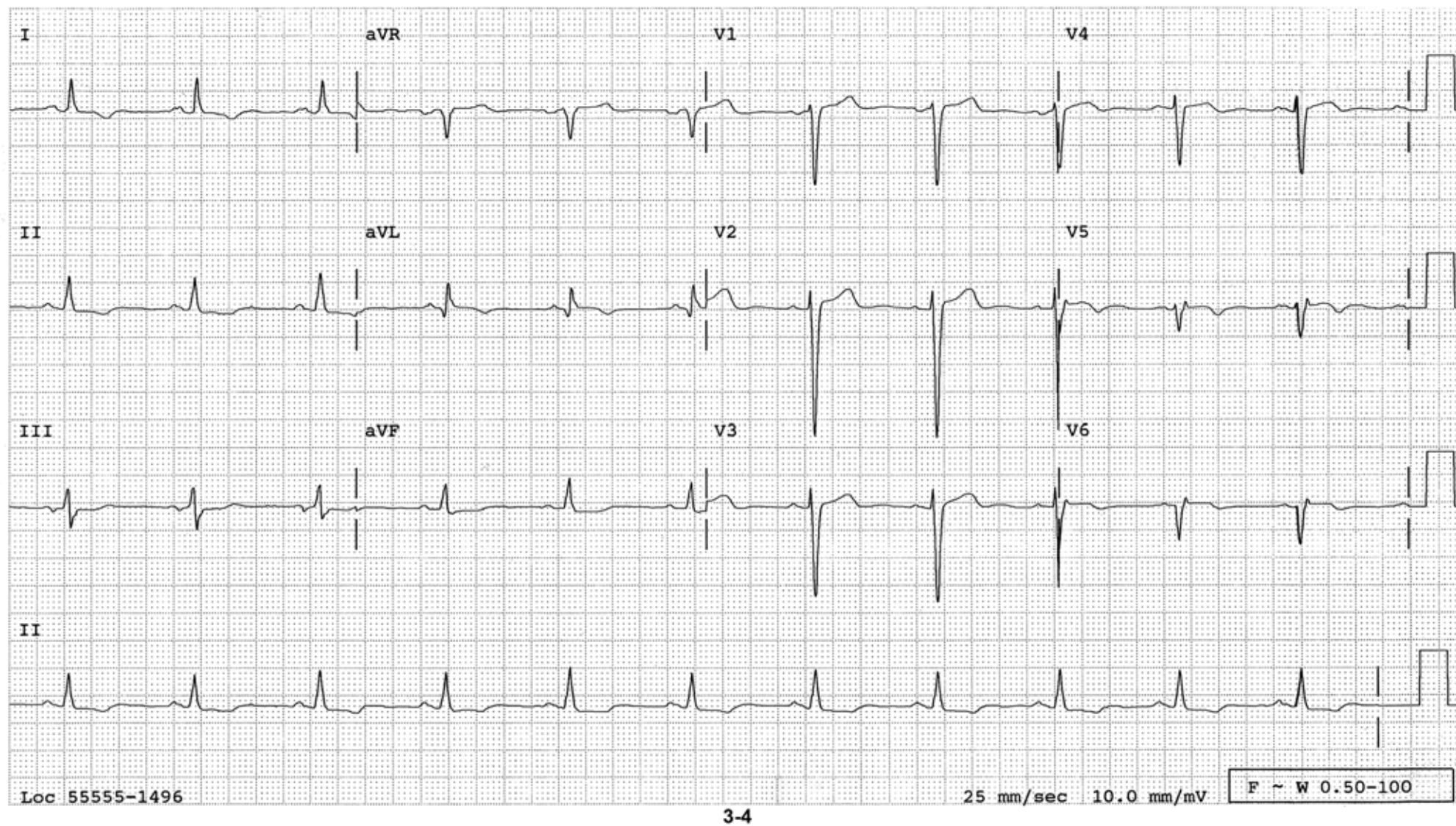
2. Anteroseptal infarct, recent



3-3

- 3-3 Atrial fibrillation with a ventricular rate of 149/minute. The last complex of the limb leads and the first two complexes in V_1 - V_3 are wide and different than other QRSs. These could be premature ventricular complexes or aberrantly conducted complexes. The RBBB pattern favors aberrant conduction. The R-R interval prior to this episode is long and this is an Ashman's phenomenon. Ashman's phenomenon states that the complex which follows a longer R-R cycle is more likely to be aberrantly conducted. This happens because the length of the refractory period is proportionally related to the preceding R-R interval and the beat which follows a longer R-R interval has an increasing chance of landing on the lengthened refractory period, thus conducting aberrantly.

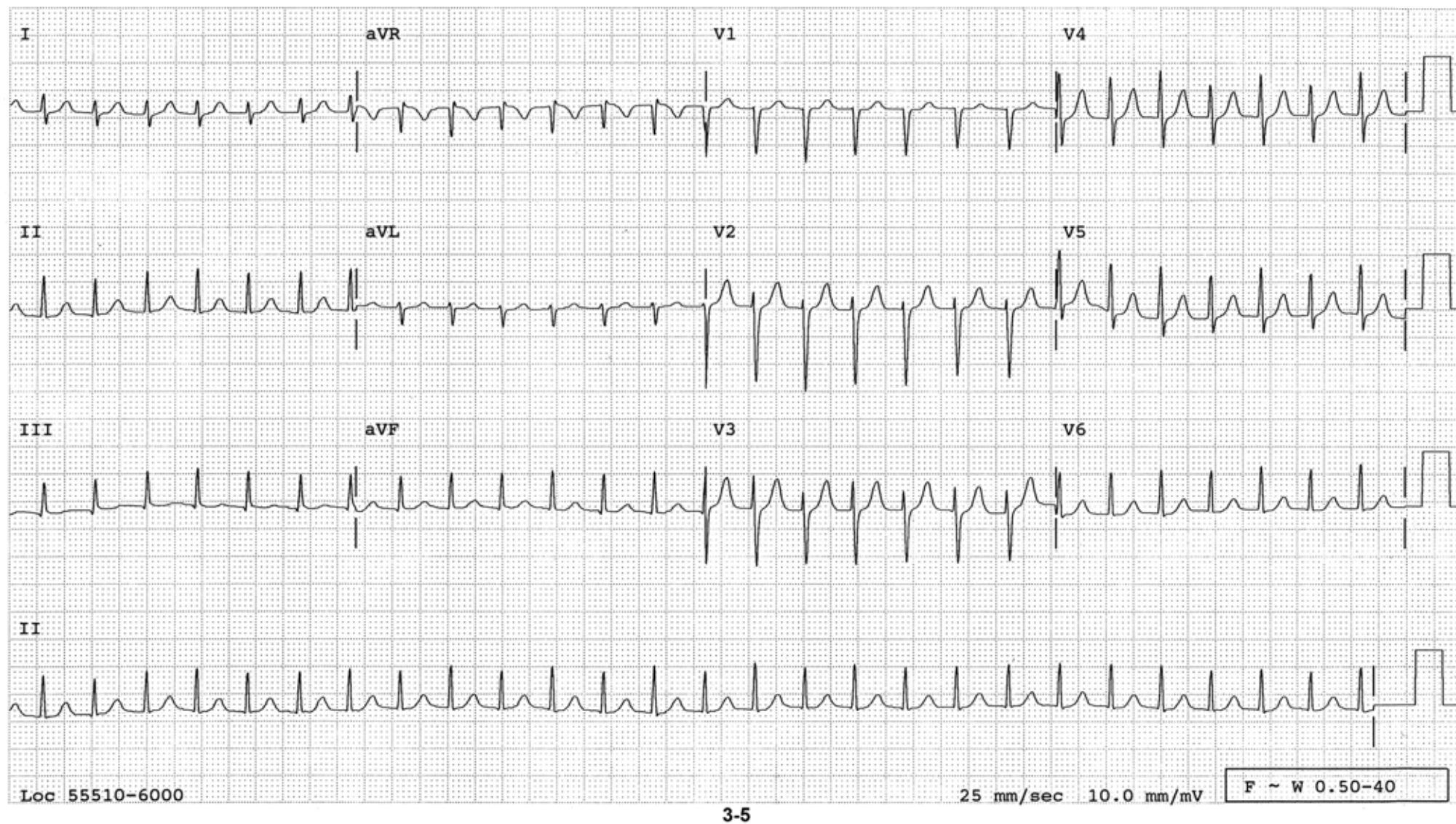
- Dx: 1. Atrial fibrillation
2. Wide complexes, most likely aberrantly conducted complexes (an Ashman's phenomenon)



3-4 Normal sinus rhythm at a rate of 67/minute. Q waves with a slight ST elevation in V₆ and aVL indicate lateral MI.

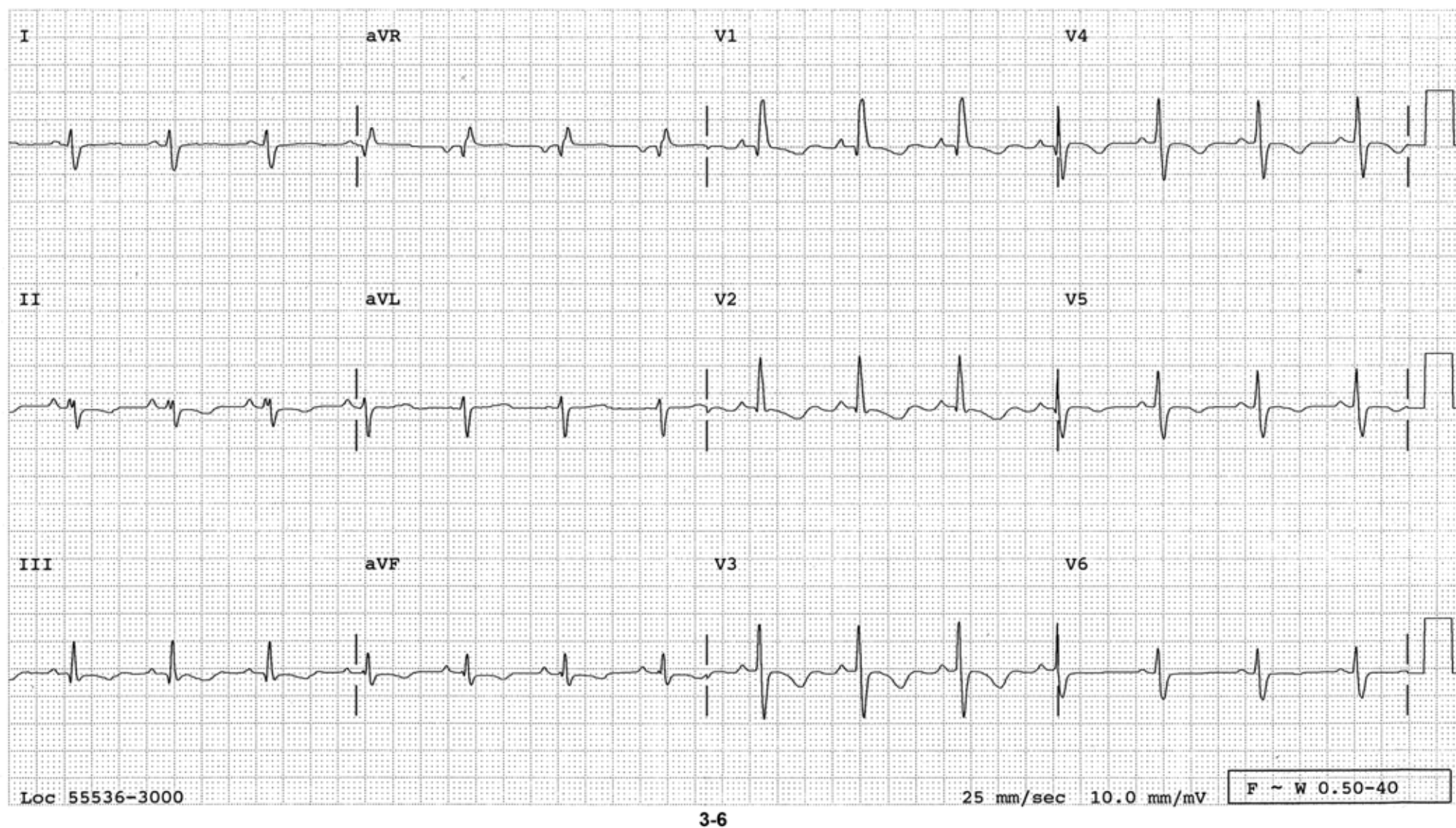
Dx: 1. NSR

2. Lateral infarct



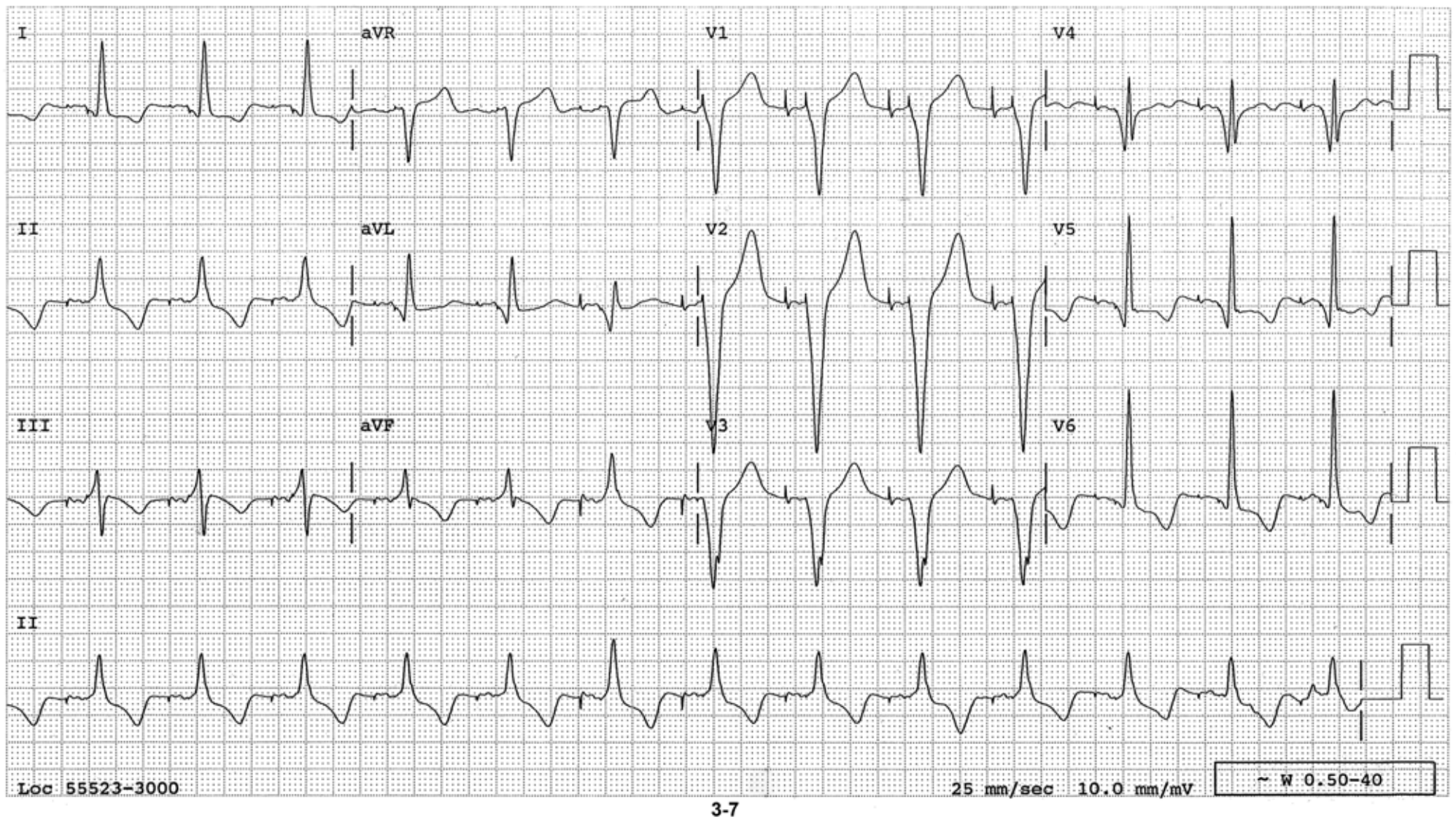
3-5 A regular narrow QRS tachycardia at a rate of 163/minute. No definite P wave can be identified. For not being able to be more specific, SVT will be called. SVT includes ectopic atrial tachycardia, AV junctional tachycardia, AV junctional reentrant tachycardia (~60% of SVTs), or AV reentrant tachycardia utilizing an accessory pathway (~30% of SVTs). These are usually indistinguishable from the surface ECG and it requires intracardiac electrogram to make the distinction.

Dx: SVT



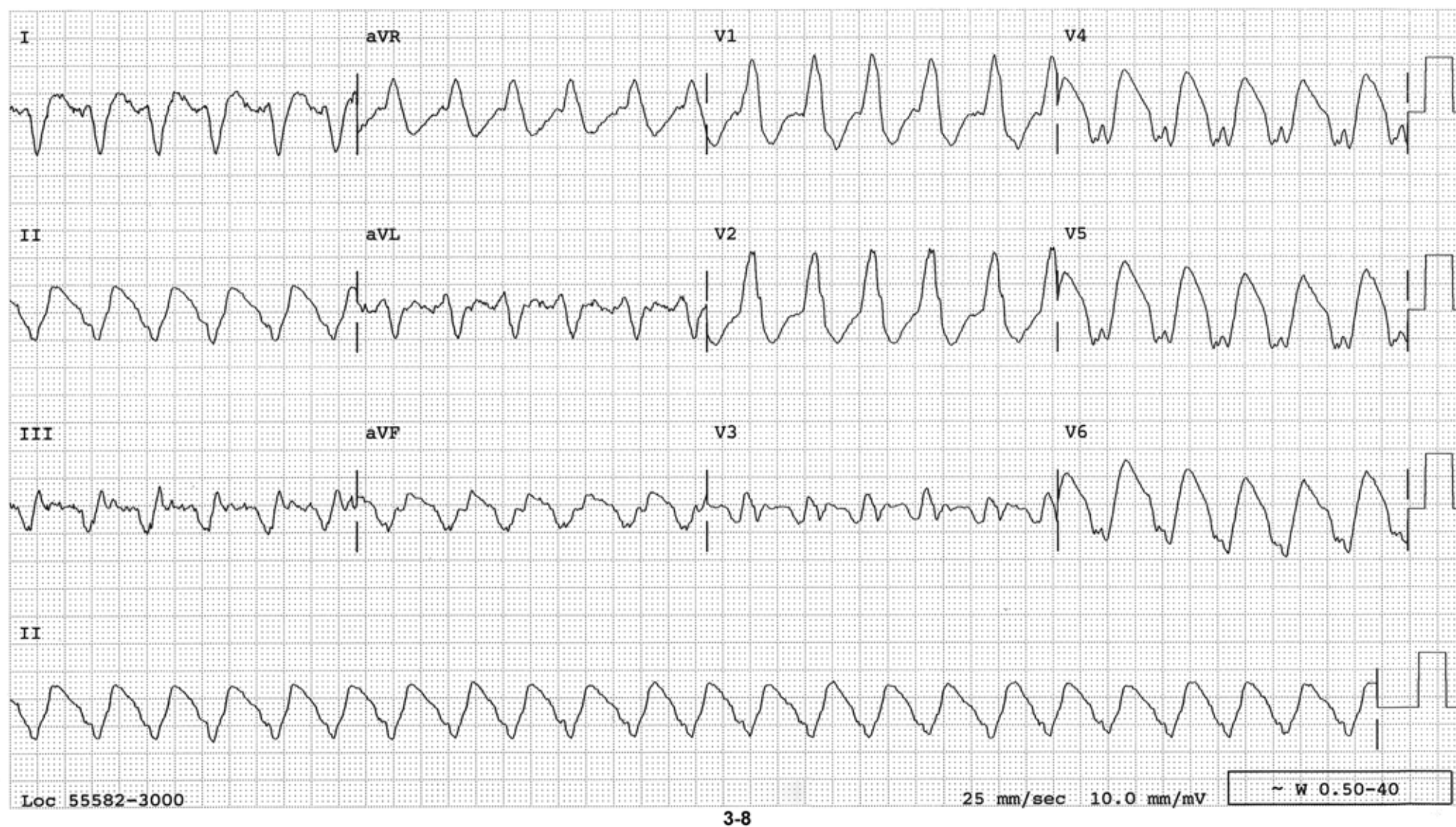
3-6 Normal sinus rhythm at a rate of 83/minute. The mean QRS axis is shifted to the right. RAD should always raise the possibility of right ventricular hypertrophy (RVH) first. A tall R wave in V_1 and deep S wave in V_6 (a hypertrophied RV being depolarized delayed causes this), ST-segment depression in the right precordial leads are all diagnostic features of RVH.

- Dx: 1. NSR
2. RVH



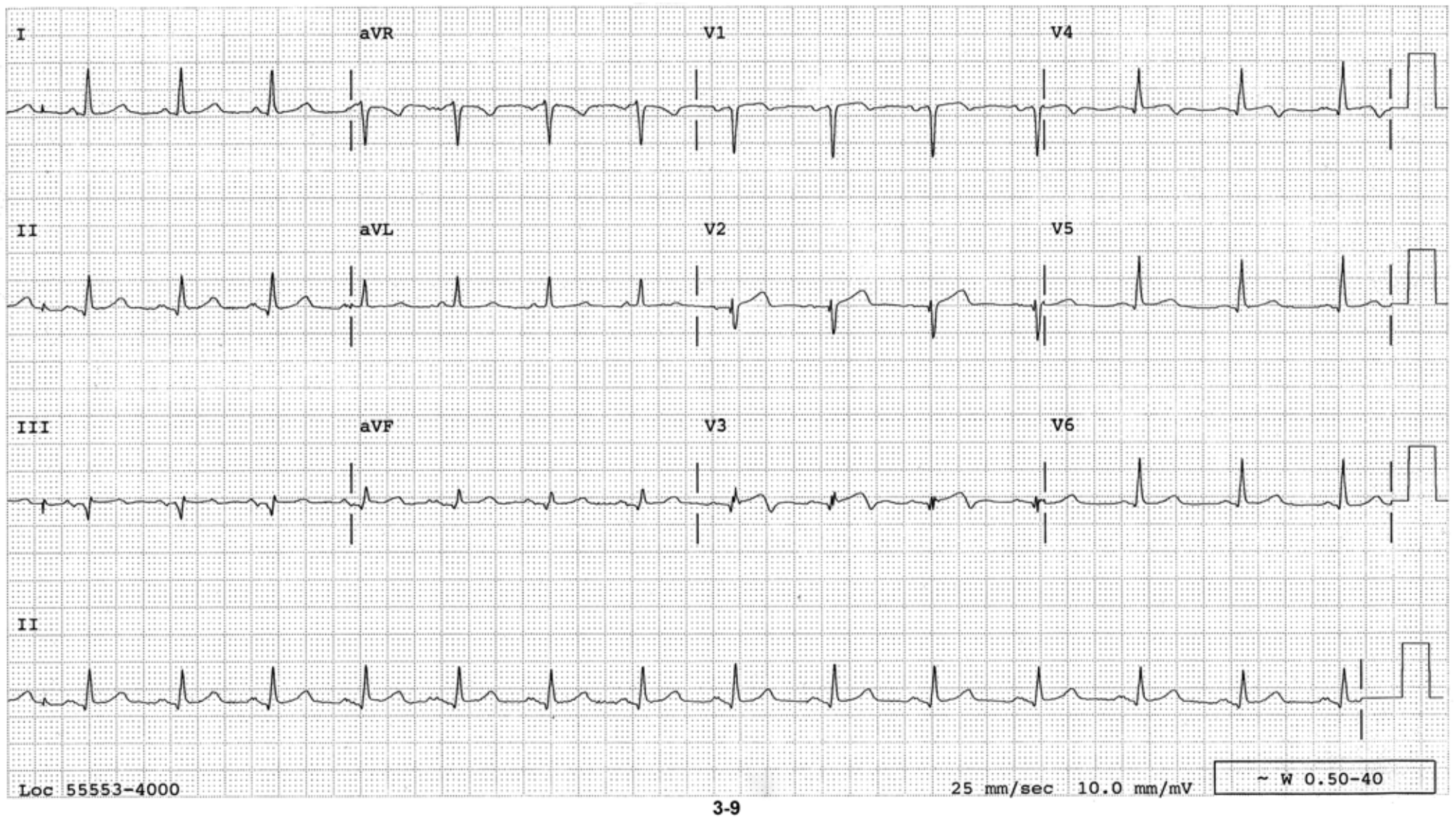
3-7 A good example of AV sequential pacemaker rhythm. The atrial pacemaker spike inducing the P wave and the ventricular pacemaker spike inducing the QRS are evident.

Dx: AV sequential paced rhythm



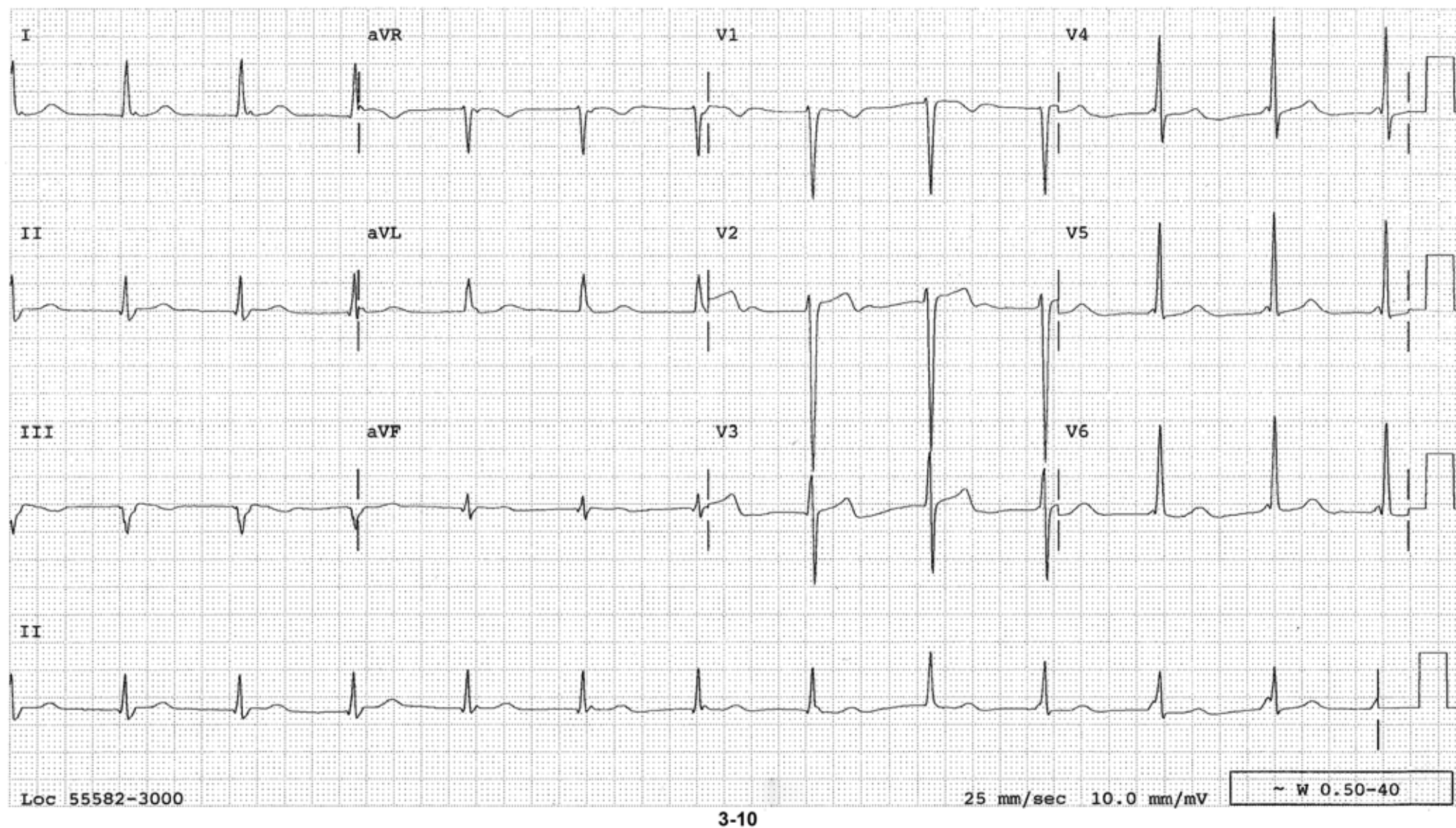
3-8 A wide QRS tachycardia at a rate of 140/minute. The QRS width measuring as wide as 224 milliseconds, monophasic R wave in V_1 and QS pattern in V_6 are highly suggestive of VT. The QR pattern in lead III raises the possibility of inferior MI.

- Dx:*
1. VT
 2. Consider old inferior MI



3-9 Normal sinus rhythm at a rate of 85/minute. The Q wave in lead III is deep and wide, diagnostic of old inferior MI. The Q waves in V_2 and V_3 combined with ST elevation and terminal T wave inversion highly suggest recent AMI.

- Dx:
1. NSR
 2. Old inferior infarct
 3. Anteroseptal infarct, recent



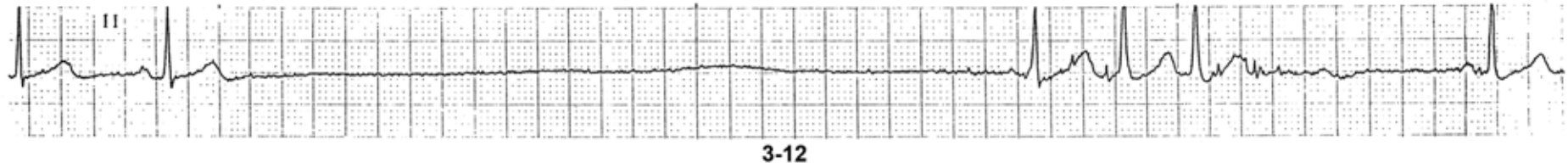
3-10 A narrow QRS rhythm at a rate of 70/minute. No P wave is present in front of the QRS initially, but they emerge during the last three QRSs indicating AV junctional rhythm with retrograde conduction to the atria during the initial four complexes (note a negative deflection at the end of the QRS early in the rhythm strip of lead II which subsequently disappears indicating that it is not part of the QRS but is a retrograde P wave), then AV dissociation subsequently. Since the junctional rate is faster than its intrinsic rate of 40-50/minute but slower than 130/minute, this rhythm will be called accelerated AV junctional rhythm. The QS pattern in lead III combined with small Q waves in leads II and aVF and mild ST elevation in III indicate recent inferior MI which may be the cause of the accelerated junctional rhythm in this patient.

- Dx: 1. Accelerated AV junctional rhythm with 1:1 retrograde conduction to the atria initially then AV dissociation
 2. Inferior infarct, recent



3-11 Atrial flutter with a variable AV conduction ratio. The flutter rate is slow at about 180/minute. Type Ia or Ic antiarrhythmic agents are well-known to slow the flutter rate to this range very easily. RBBB and abnormal left axis deviation indicate BIFB (RBBB and LAFB). The third and fourth complexes from the end are different and wide and most likely ventricular premature complexes.

- Dx:*
1. Atrial flutter with variable AV conduction ratio
 2. BIFB
 3. PVCs

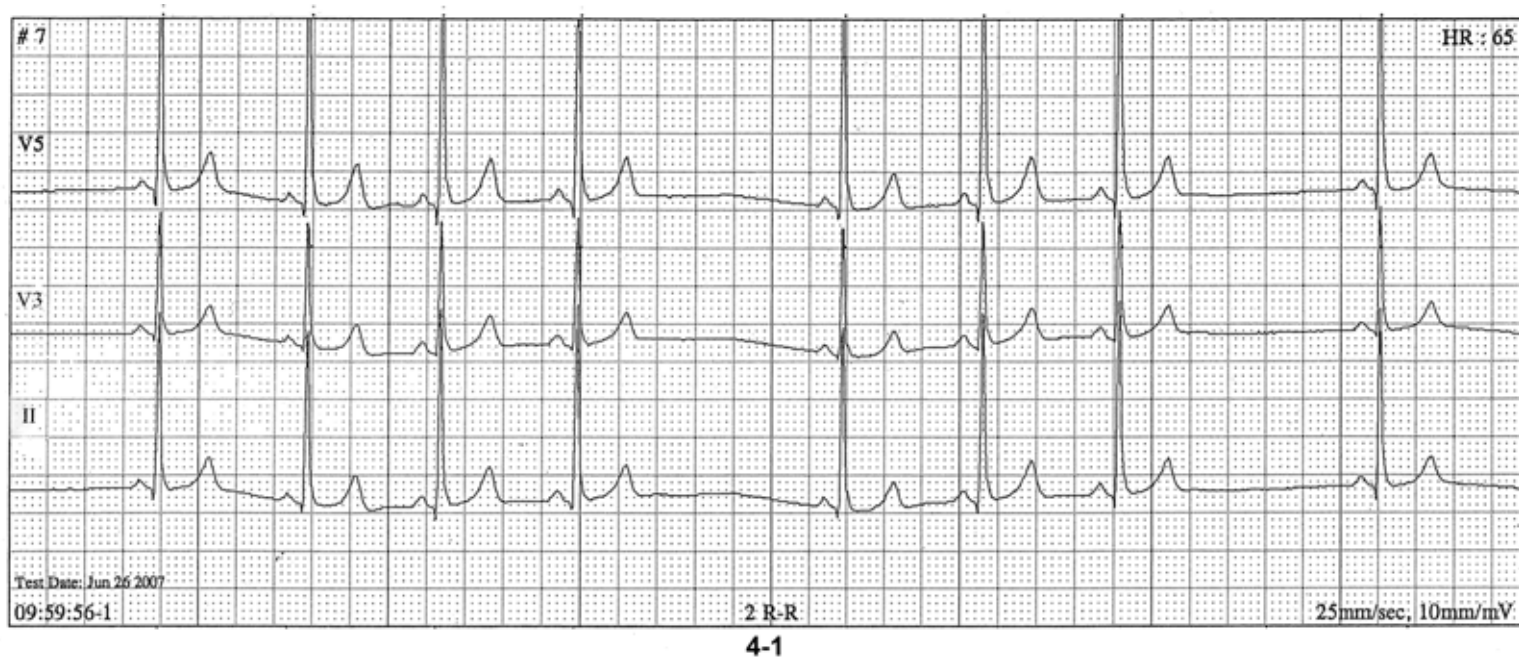


3-12 A long “pause” due to a transient vagotonic state such as from:

1. Neurocardiogenic syncope (vagovagal syncope)
2. Spontaneously or during carotid sinus massage in a patient with hypersensitive carotid sinus syndrome
3. Paroxysms of cough
4. Micturition
5. Retching, vomiting
6. Tracheal suctioning, etc.

Dx: A long pause (cardioinhibitory response) during a vagotonic state in various situations. “Cardioinhibitory” because not only the sinus node is suppressed but also the AV nodal and ventricular escape mechanisms are suppressed.

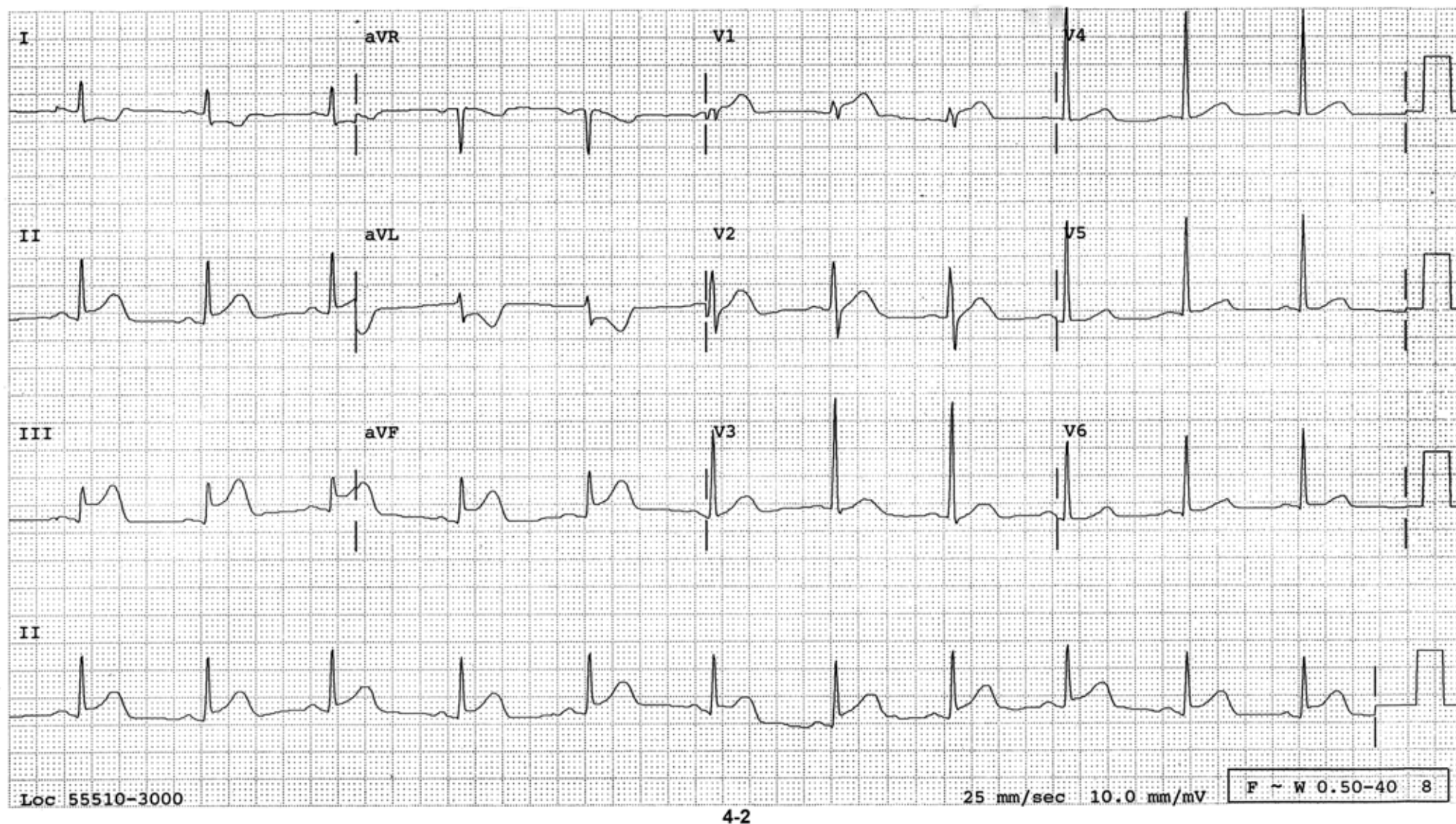
SECTION 4



- 4-1 Regular sinus rhythm is interrupted by sudden pauses. During the pauses, there are no extra P waves to consider either AV block or nonconducted atrial premature impulses. The pauses are equal to two R-R intervals and are typical examples of block between the sinus node and the atrium: sinoatrial (SA) block, Type II. If it were Type I, the pauses would have been shorter than two R-R intervals.

Sinoatrial block is a form of sinus node dysfunction and is often due to medications. If it is intrinsic, it will be called sick sinus syndrome. Only if the patient is symptomatic from the pauses an electronic pacemaker will be indicated. If this patient is asymptomatic, the patient will be followed without a pacemaker.

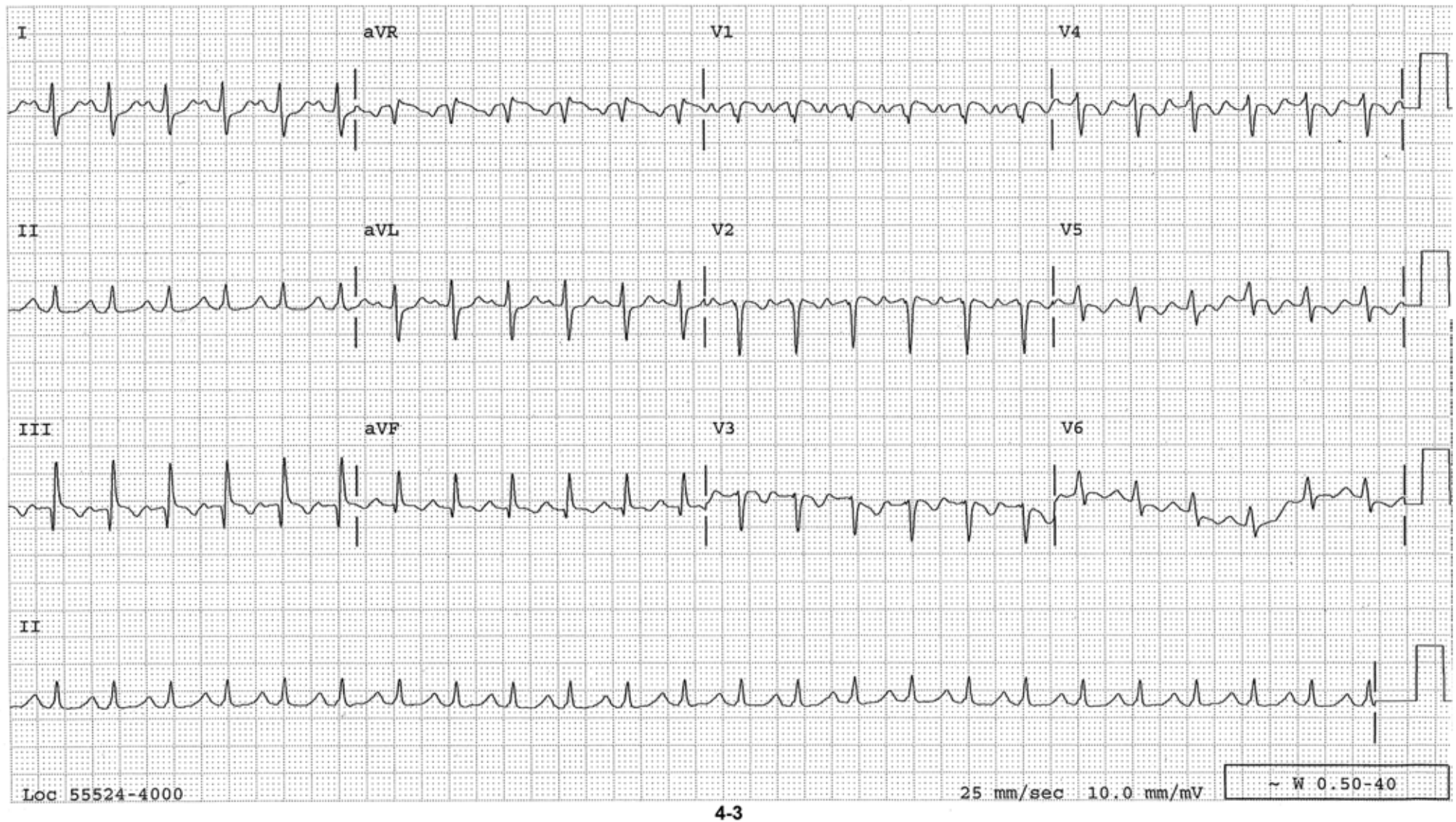
Dx: 1. Sinoatrial block, Type II



4-2 Normal sinus rhythm at a rate of 68/minute. ST elevation in the inferior leads is diagnostic of inferior STEMI; In inferior STEMI, the ST-segment is always depressed reciprocally in aVL. If it is depressed in lead I as well, it means the ST vector is pointed down and to the right. Why to the right? Because the RV is involved, which means the culprit lesion is in the proximal right coronary artery (RCA). The RV involvement is also manifested by ST elevation in V_1 in this patient. R waves in V_1 - V_3 are tall indicating posterior wall involvement as well. In acute posterior MI, the ST-segment is horizontally depressed in these leads, but that is not the case in this patient. Most likely it is cancelled out by the ST elevation from RV involvement.

Dx: 1. NSR

2. Acute inferoposterior infarct with RV involvement due to proximal RCA occlusion



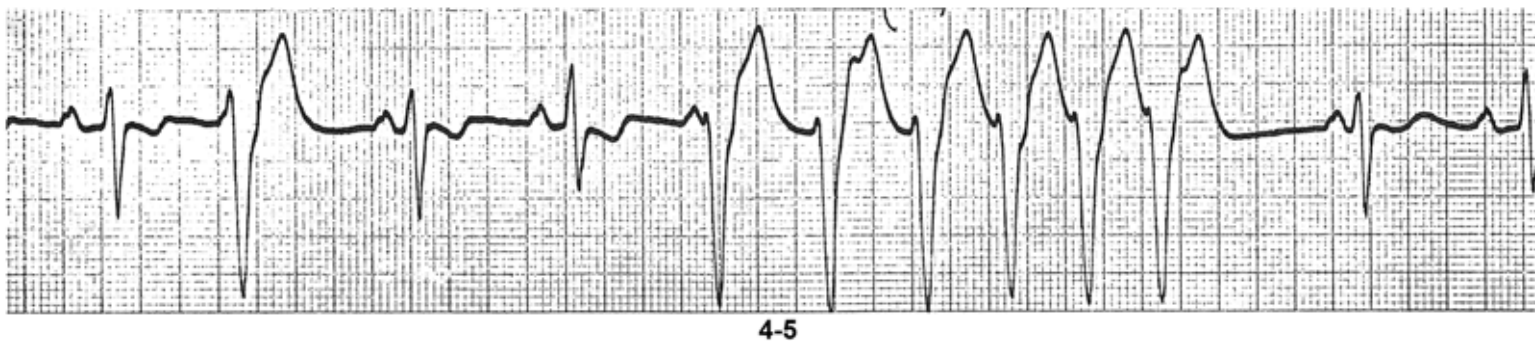
4-3 Sinus tachycardia at 144/minute. There is an S wave in lead I, Q wave in lead III and T wave inversion in lead III, hence called $S_1Q_3T_3$ pattern. This $S_1Q_3T_3$ pattern combined with sinus tachycardia is highly suggestive of pulmonary embolism. T wave inversion in V_1 - V_3 is consistent with RV strain, also supporting acute RV overload. In $S_1Q_3T_3$ pattern, unlike in inferior infarction, the Q wave is not wide.

Dx: $S_1Q_3T_3$ pattern with sinus tachycardia, highly suggestive of pulmonary embolism



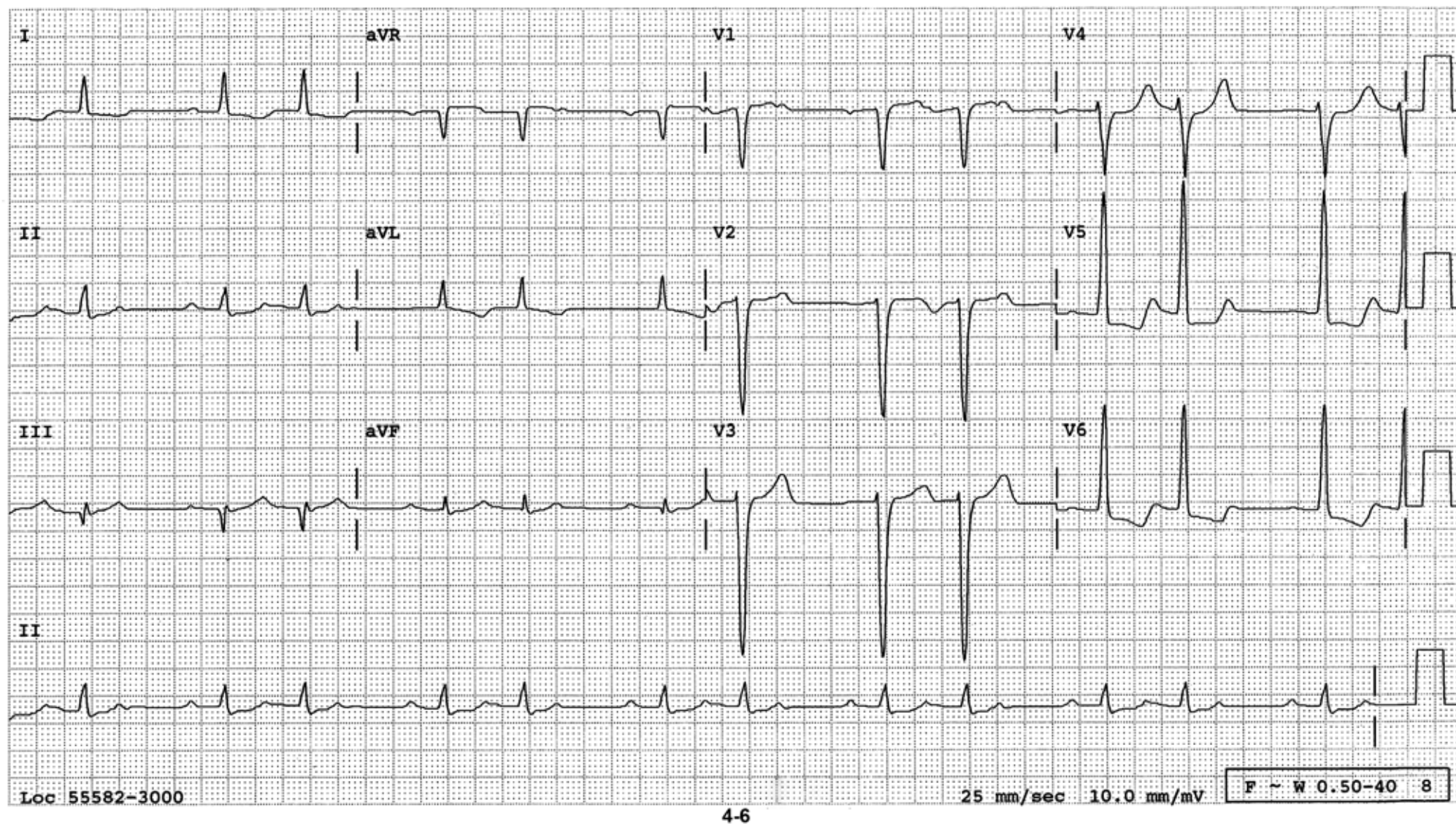
4-4 Normal sinus rhythm at a rate of 94/minute. The QRS voltage is abnormally low (QRS voltage does not exceed 5 mm in the limb leads). The Q wave in lead III, even though shallow, is significant considering the total amplitude of the QRS. It is indeed wide enough (more than 40 milliseconds). This, combined with slight ST elevation, indicates acute inferior MI. ST-segment is depressed in lead I as well as in aVL indicating RV involvement from proximal RCA occlusion. The R waves are not progressing appropriately and may reflect old AMI as well.

- Dx:
1. NSR
 2. Low QRS voltage
 3. Acute inferior infarct with RV involvement
 4. Probable old anteroseptal infarct



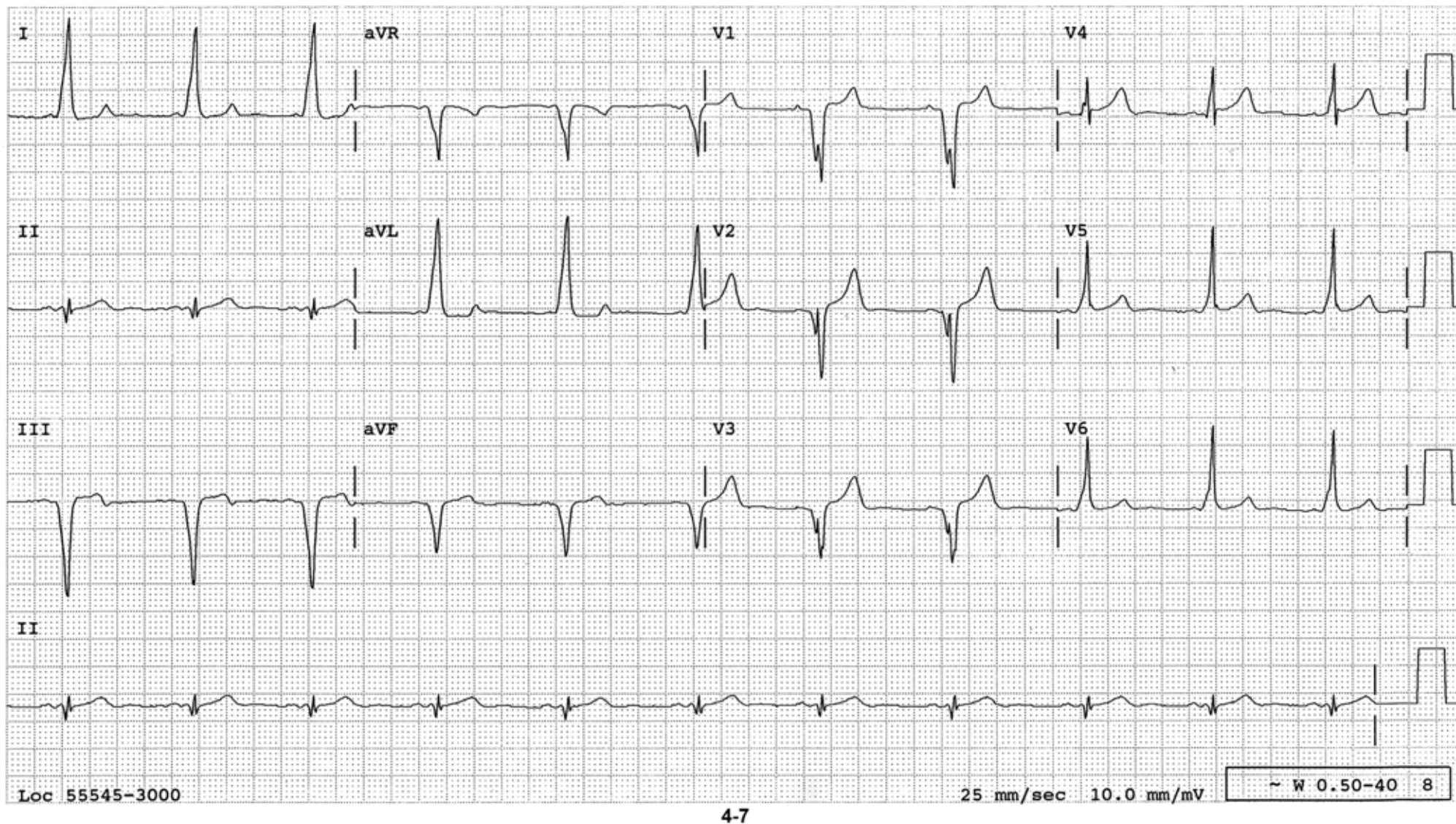
4-5 Normal sinus rhythm at a rate of 74/minute. The second QRS is a ventricular premature complex. The fifth QRS again is wide and occurs slightly prematurely. Even though there is a P wave in front of it, the P-R interval is too short for the P to result in the QRS. Hence, it is a ventricular premature complex and this run of wide QRS tachycardia is VT. A slightly irregular R-R interval is perfectly compatible with VT.

- Dx:*
1. NSR
 2. A PVC and a six beat run of VT



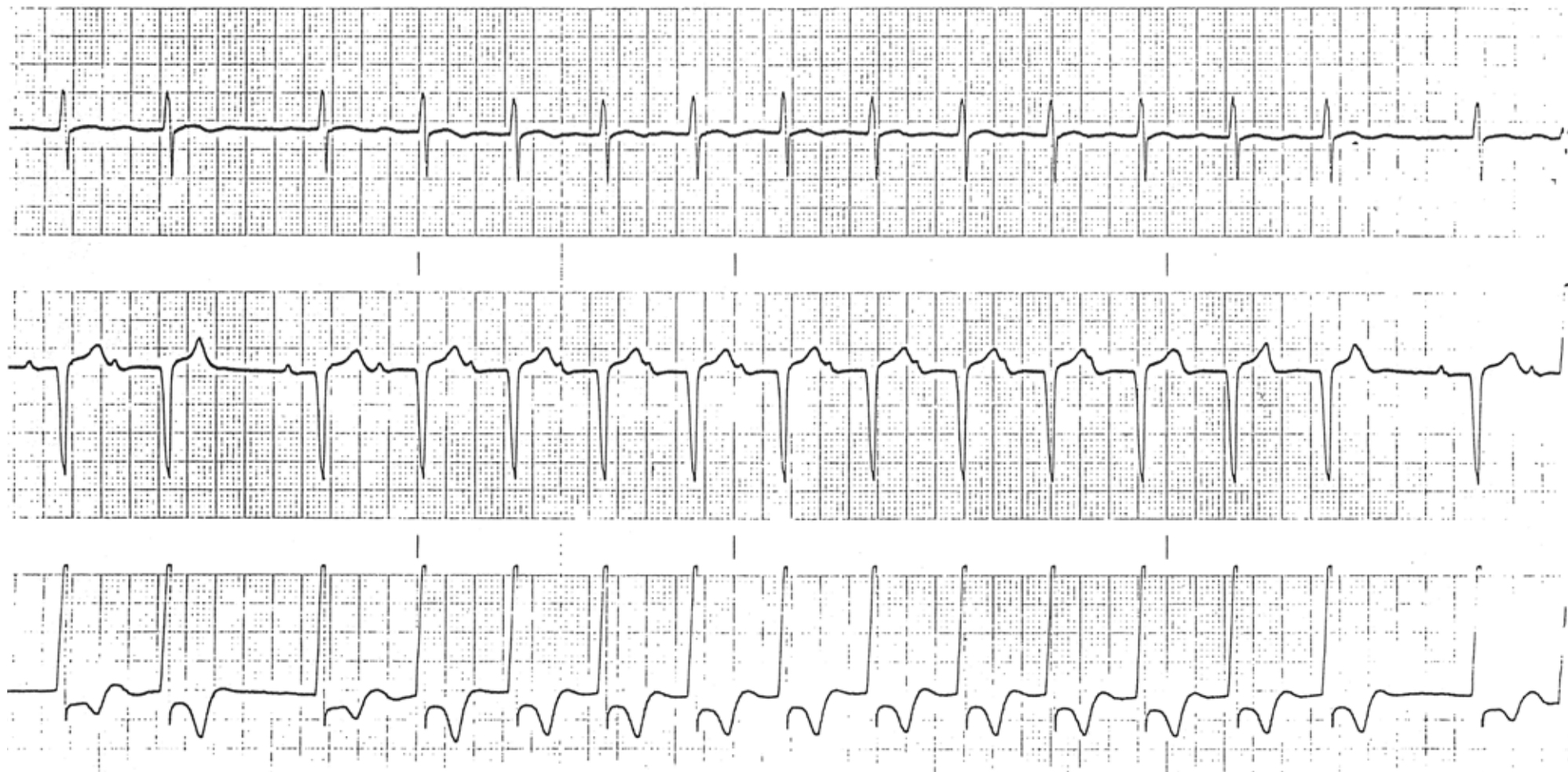
4-6 P waves occur regularly at a rate of 110/minute. QRSs are paired. The rhythm strip reveals that the P-R interval of the second of the paired complexes is longer than the first, and the third P wave which is superimposed upon the T wave is blocked; a typical 3:2 AV Wenckebach phenomenon. The Q wave in lead III is deep and wide enough suggesting old inferior MI. Voltage criteria and ST-T changes for LVH are present.

- Dx:*
1. Sinus tachycardia
 2. 3:2 AV Wenckebach phenomenon
 3. Old inferior infarct
 4. LVH



4-7 Normal sinus rhythm at a rate of 65/minute. P-R interval is short and the upstroke of QRSs is slurred (delta wave) reflecting Wolff-Parkinson-White syndrome. The findings in the right precordial leads raise the possibility of antero-septal infarction and the inferior leads suggesting inferior infarct, but this is an example of a posteriorly and superiorly directed delta wave, resulting in inverted delta waves in these leads simulating these infarcts.

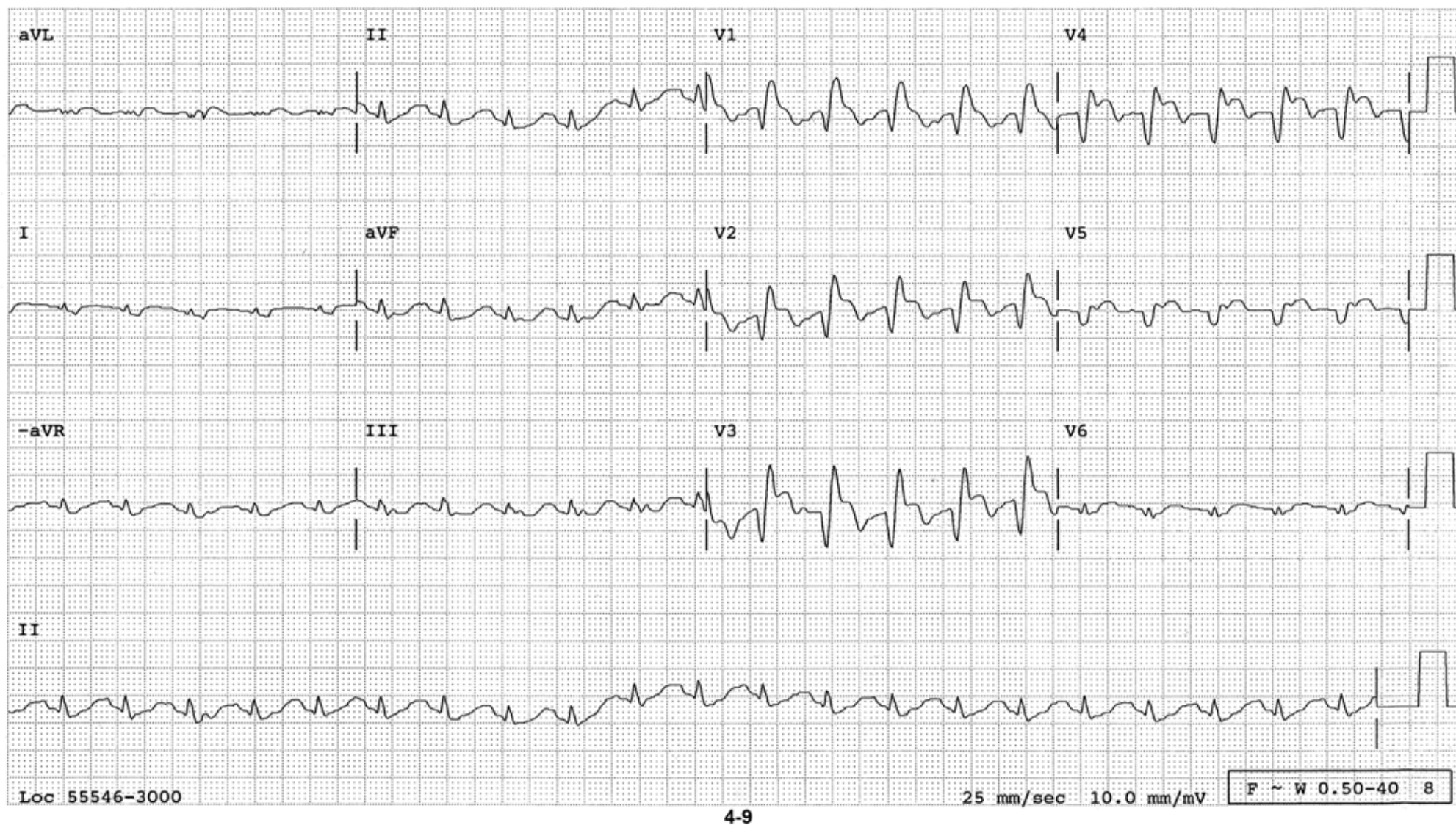
Dx: Wolff-Parkinson-White syndrome simulating antero-septal and inferior infarct



4-8

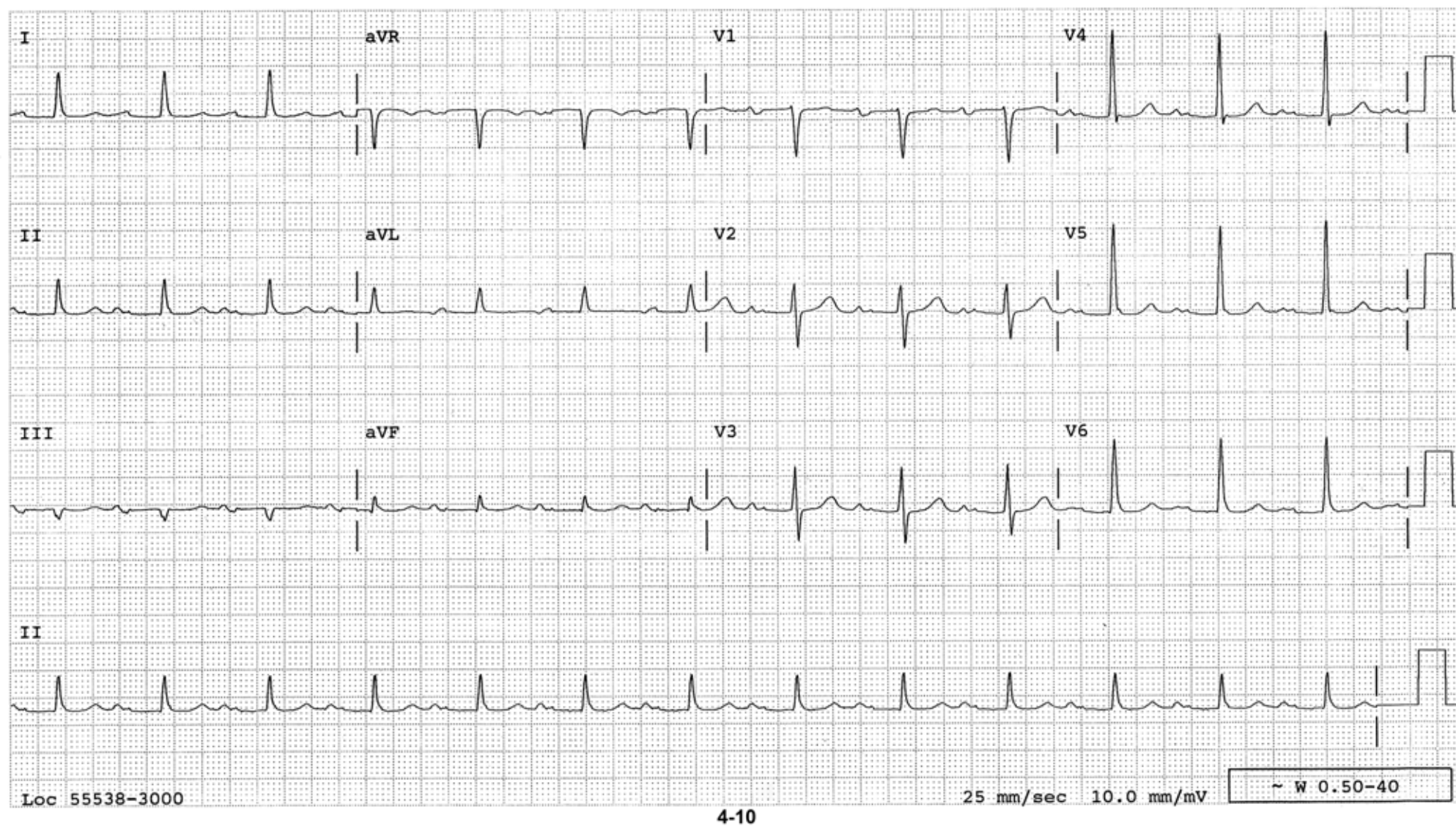
4-8 Rhythm strips reveal sinus rhythm with a progressive lengthening P-R interval until eventually a P wave is blocked. This is clearly appreciated in the middle strip but hardly at all in other strips. The P wave is easily recognizable in the early part of the Wenckebach cycle and when the P wave is superimposed on the T wave, it becomes obscured.

Dx: Type I 2° AV block (AV Wenckebach phenomenon)



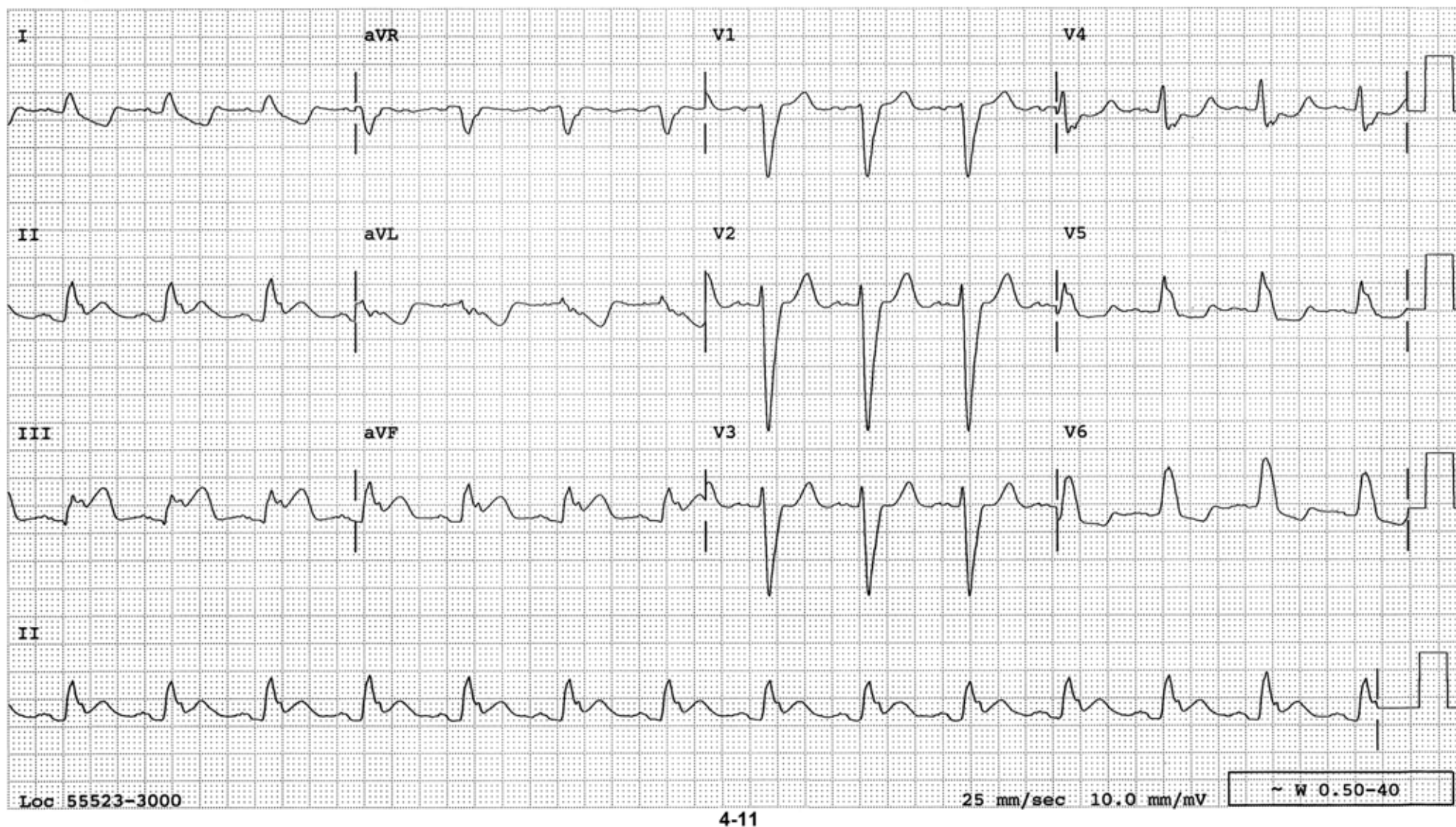
4-9 Sinus tachycardia at a rate of 129/minute. The QRS voltage is low in the limb leads. A QR pattern with ST elevation in the precordial leads indicates acute AMI as well as RBBB. The R wave of the QR in the right precordial leads is actually the R' of rsR' of RBBB remaining while the r of rsR' is taken away by the anteroseptal MI.

- Dx:*
1. Sinus tachycardia
 2. Low QRS voltage
 3. Acute anterior infarct
 4. RBBB



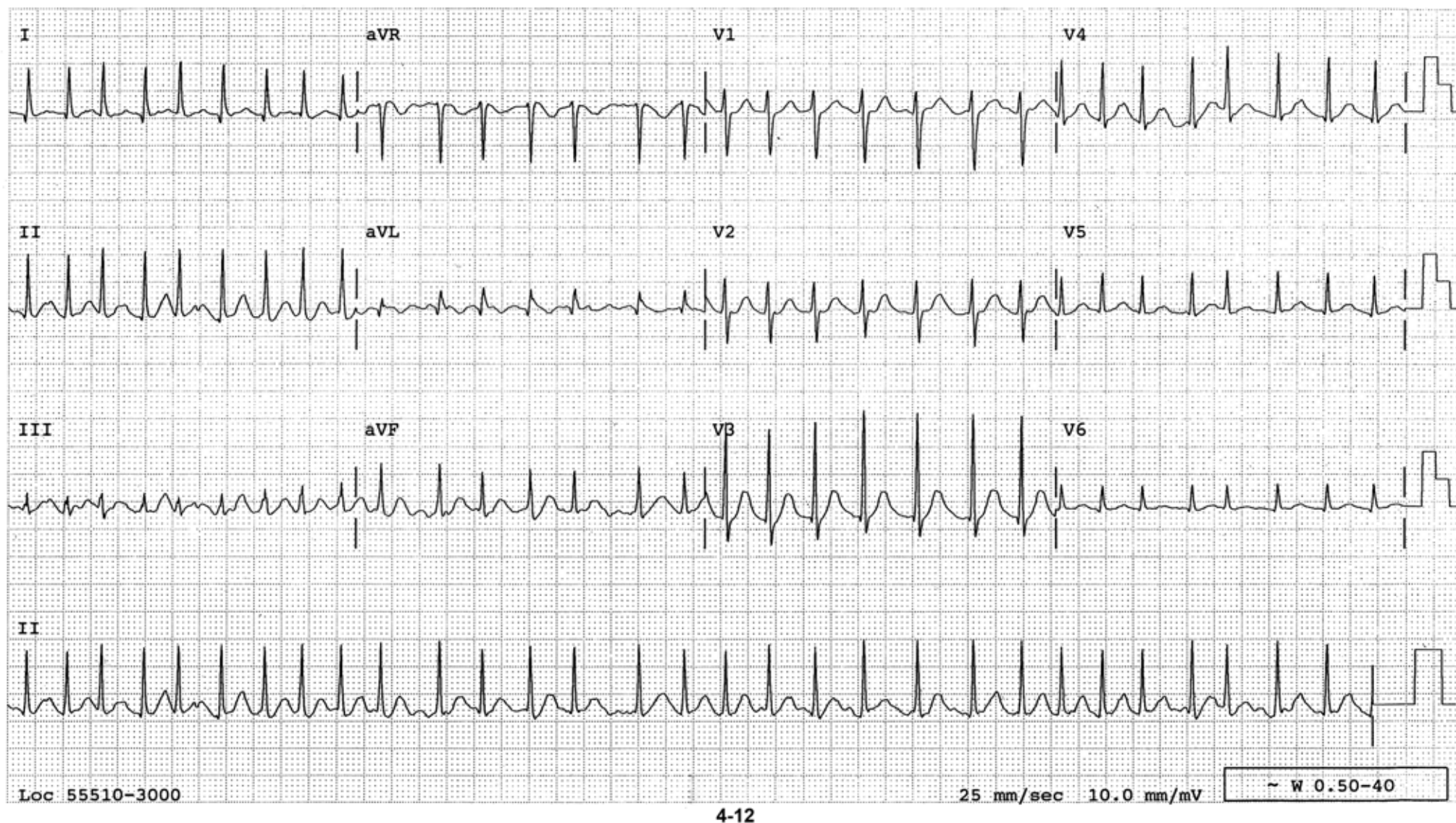
4-10 Normal sinus rhythm at a rate of 78/minute. The P-R interval is prolonged to 340 m/second. However, every P wave results in a QRS indicating 1° AV block. The P wave is mostly negative in V₁ suggesting left atrial abnormality.

- Dx:
1. NSR
 2. 1° AV block
 3. LAE



4-11 Regular rhythm at 85/minute. QRSs are wide (160 milliseconds) but there is a P wave in front of each QRS indicating the rhythm is sinus. The wide QRS is made of monophasic R wave in leads I and V_5 - V_6 , and is mostly negative in V_1 - V_3 ; typical of LBBB. In the inferior leads, there are ST changes concordant with the major component of the QRS (QRS is upright and ST-segment is elevated), and are diagnostic of acute MI. Thus, acute MI can be recognized even with LBBB if there is a concordant ST change, i.e. ST-segment deviated to the same direction as the major component of the QRS. Uncomplicated LBBB just does not do that.

- Dx:
1. NSR
 2. LBBB
 3. Inferior STEMI

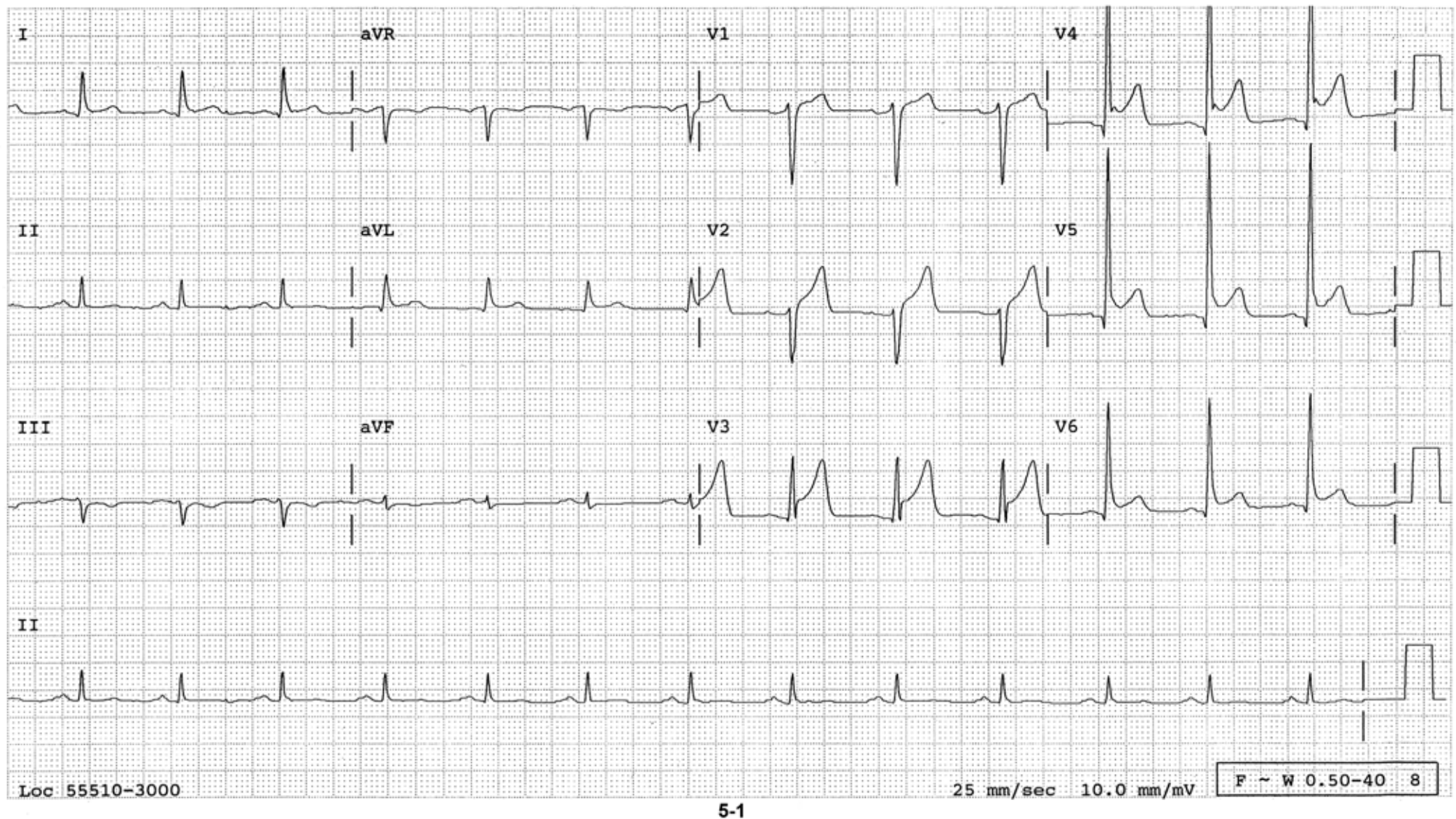


4-12

4-12 Irregularly irregular rhythm at a rate of 187/minute. No distinctive P waves can be identified and this is a good example of atrial fibrillation. Sometimes, when the fibrillatory wave occurs just at the right time in front of the QRS it may simulate a sinus P wave as happened with the eleventh or the second complex from the end in the rhythm strip.

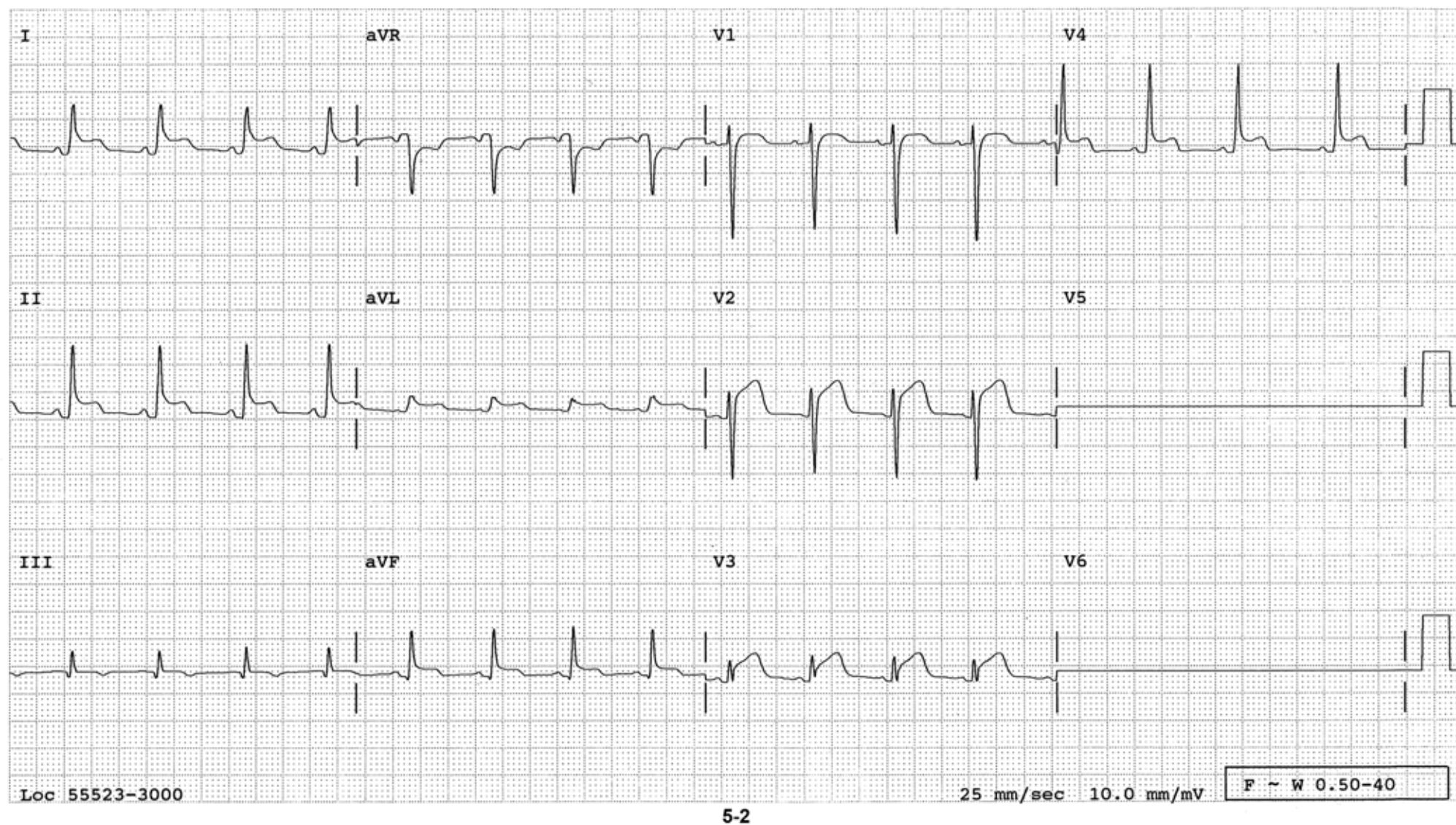
Dx: Atrial fibrillation with a ventricular rate of 189/minute

SECTION 5



5-1 Normal sinus rhythm at a rate of 80/minute. QRS voltage for LVH is present. There is about 4 mm ST elevation in V_3 - V_4 , and to a lesser degree in other precordial leads. The notching in the junction in V_4 and upward concavity of the ST-segment are all diagnostic of early repolarization pattern as a normal variant. The PR-segment is slightly depressed which is also part of this condition. Sometimes these findings may be mistaken for an acute MI or pericarditis.

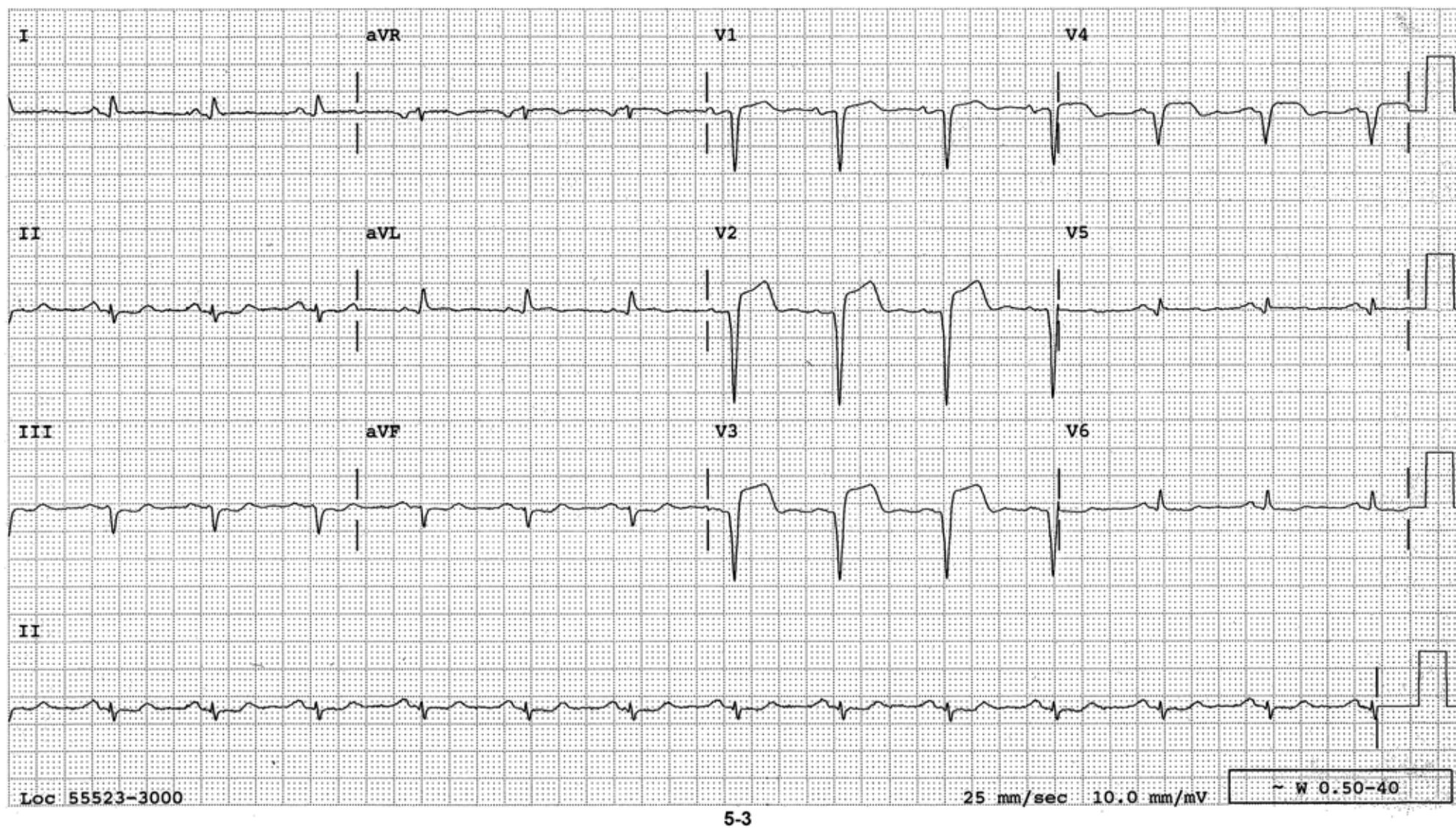
- Dx:
1. NSR
 2. LVH by voltage
 3. Early repolarization pattern as a normal variant



5-2 Normal sinus rhythm at a rate of 98/minute. Diffuse ST elevation combined with PR-segment depression is consistent with an acute pericarditis. PR-segment depression is the atrial counterpart of the ST-segment elevation. Note that there is no notching in the junction and the ST-segment is either horizontal or convex. The findings in the right precordial leads alone certainly suggest AMI. The fact that the ST-segment elevation involves both the precordial and limb leads, that is, more than one vascular territory simultaneously would be very unusual for an acute MI (V_5 and V_6 leads are off).

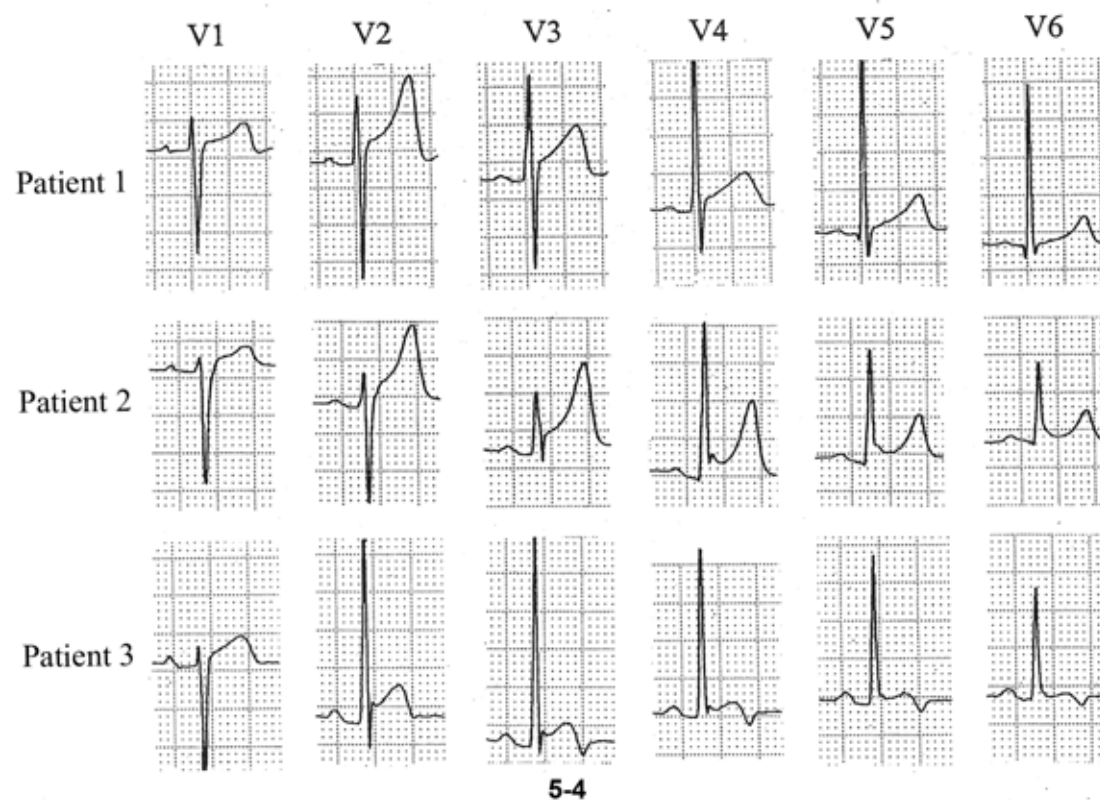
Dx: 1. NSR

2. Diffuse ST elevation and PR-segment depression, very good for acute pericarditis



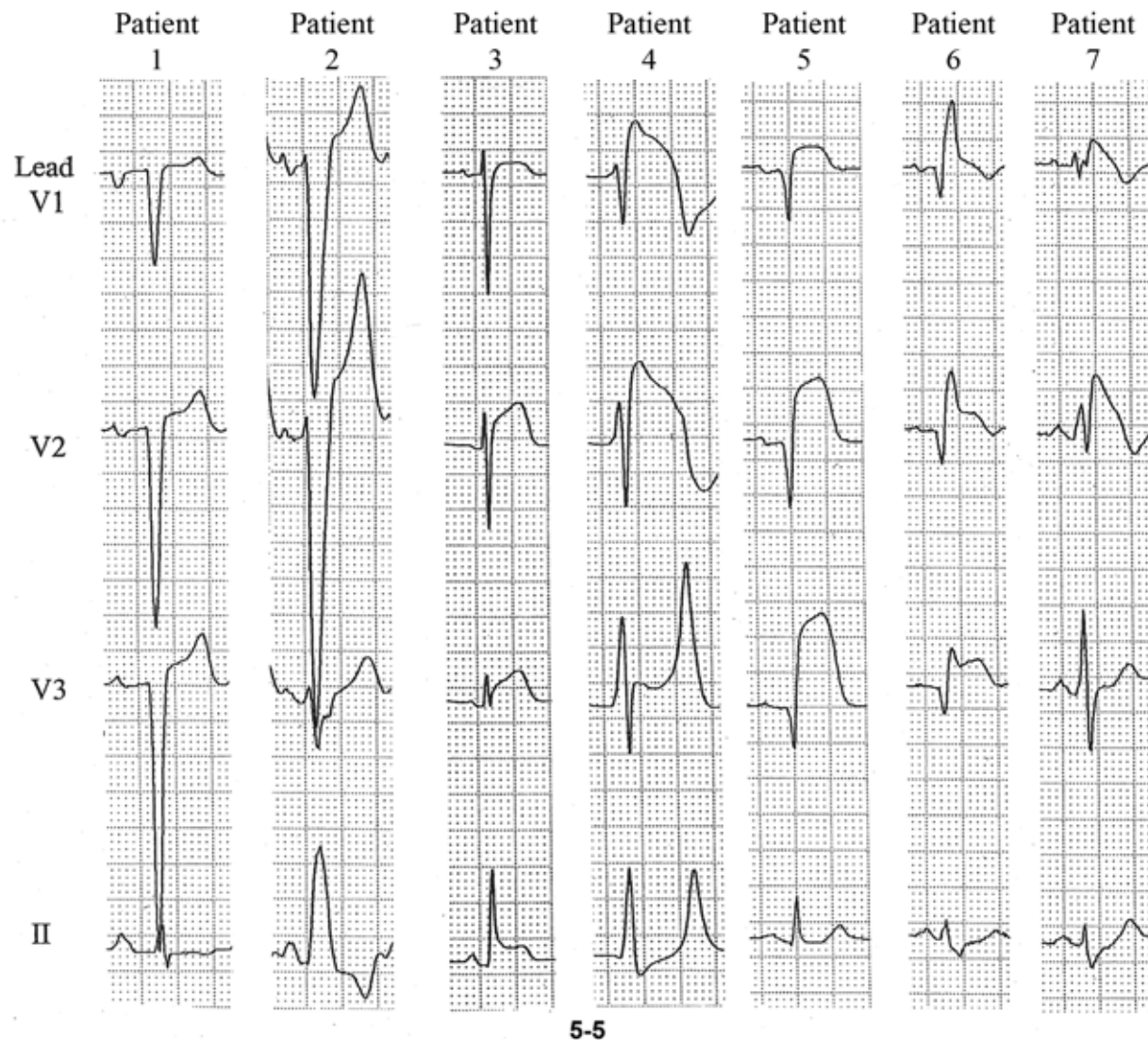
5-3 Normal sinus rhythm at a rate of 79/minute. The QRS voltage is low in the limb leads and there is an abnormal left axis deviation consistent with LAFB with or without previous MI. The striking features are Q waves with significant ST elevation in V₁ through V₅ reflecting extensive acute anterior MI.

- Dx:*
1. NSR
 2. LAFB with or without old inferior infarct
 3. Acute anterior infarct



5-4 ST elevation in the precordial leads from three different Patients.

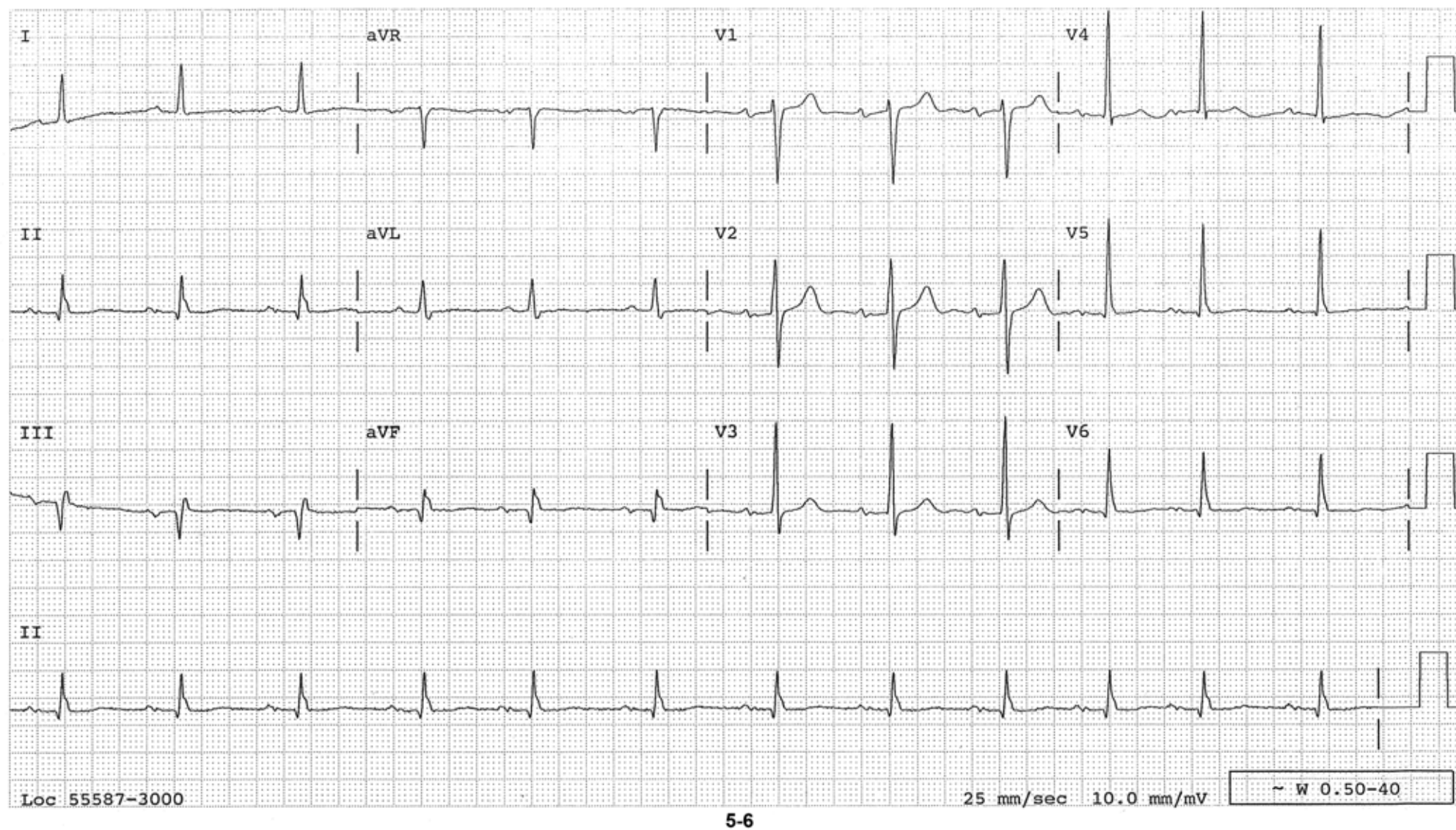
1. Normal ST elevation. Over 90% of healthy young men have 1–3 mm ST elevation in the right precordia leads normally. The ST-segment is concave. The deeper the S wave, the more the ST elevation.
2. Early repolarization pattern as a normal variant. There is a notch at the junction (usually in V₄). The T waves are upright and tall.
3. ST elevation of “the other” normal variant. This entity is still different from the early repolarization pattern in that the T waves are inverted. Seen almost exclusively in young black men; often mistaken for acute pericarditis or infarct. The Q-T interval tends to be short.



5-5

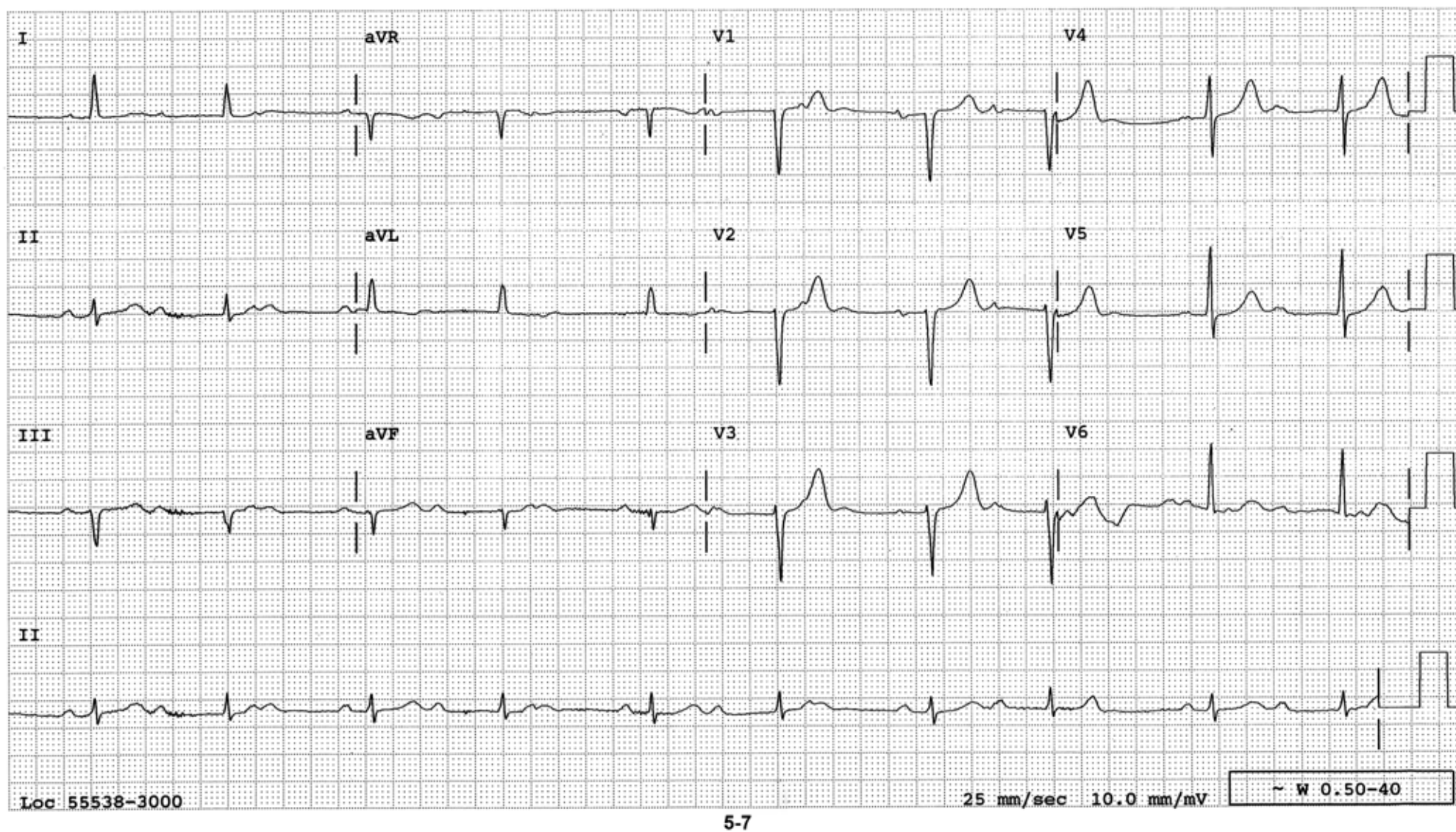
5-5 Lead V_1 - V_3 and II from 7 different patients who all have ST elevation.

1. LVH
2. LBBB
3. Pericarditis. Only tracing with ST elevation also in lead II and significant PR-segment depression
4. Hyperkalemia with pseudoinfarction pattern. Note tall, pointed, tented and narrow T wave in V_3
5. Acute AMI. Note smooth ST-segment with no second "rabbit ear" sticking out
6. Acute AMI and RBBB. Note the second "rabbit ear" (R') sticking out. Note also distinct transition between the downstroke of R' and the beginning of the ST-segment
7. Brugada syndrome. The elevated ST-segment begins from the top of the R' and is downsloping, ending in a negative T wave. Unlike in patient 6, there is no distinct transition between the downstroke of R' and the beginning of the ST-segment



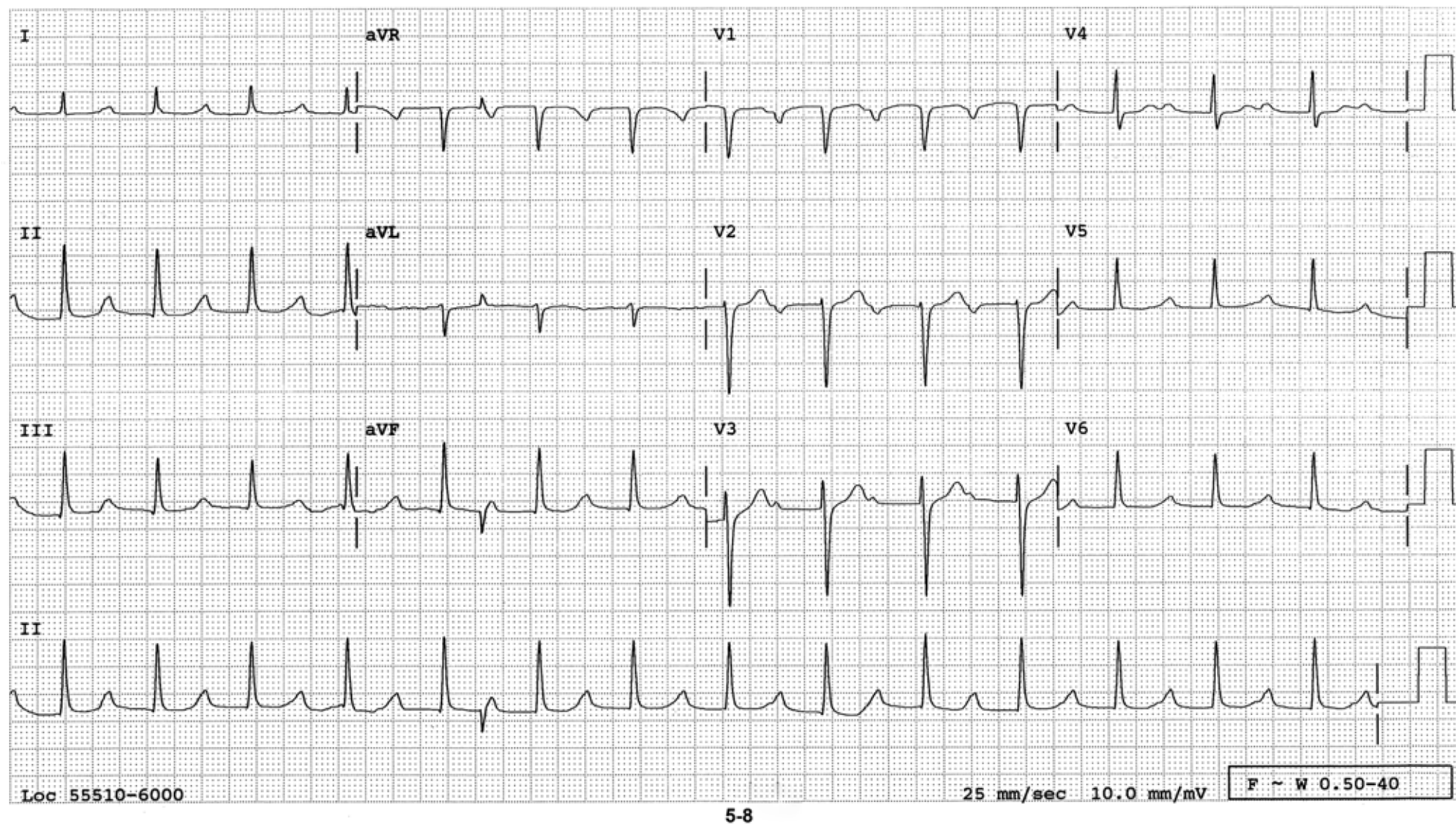
5-6 Sinus rhythm at a rate of 72/minute. The P-R interval is prolonged to 220 millisecond indicating 1° AV block. The Q wave in lead III is deep and wide, diagnostic of old inferior infarct.

- Dx:*
1. Sinus rhythm
 2. 1° AV block
 3. Old inferior infarct



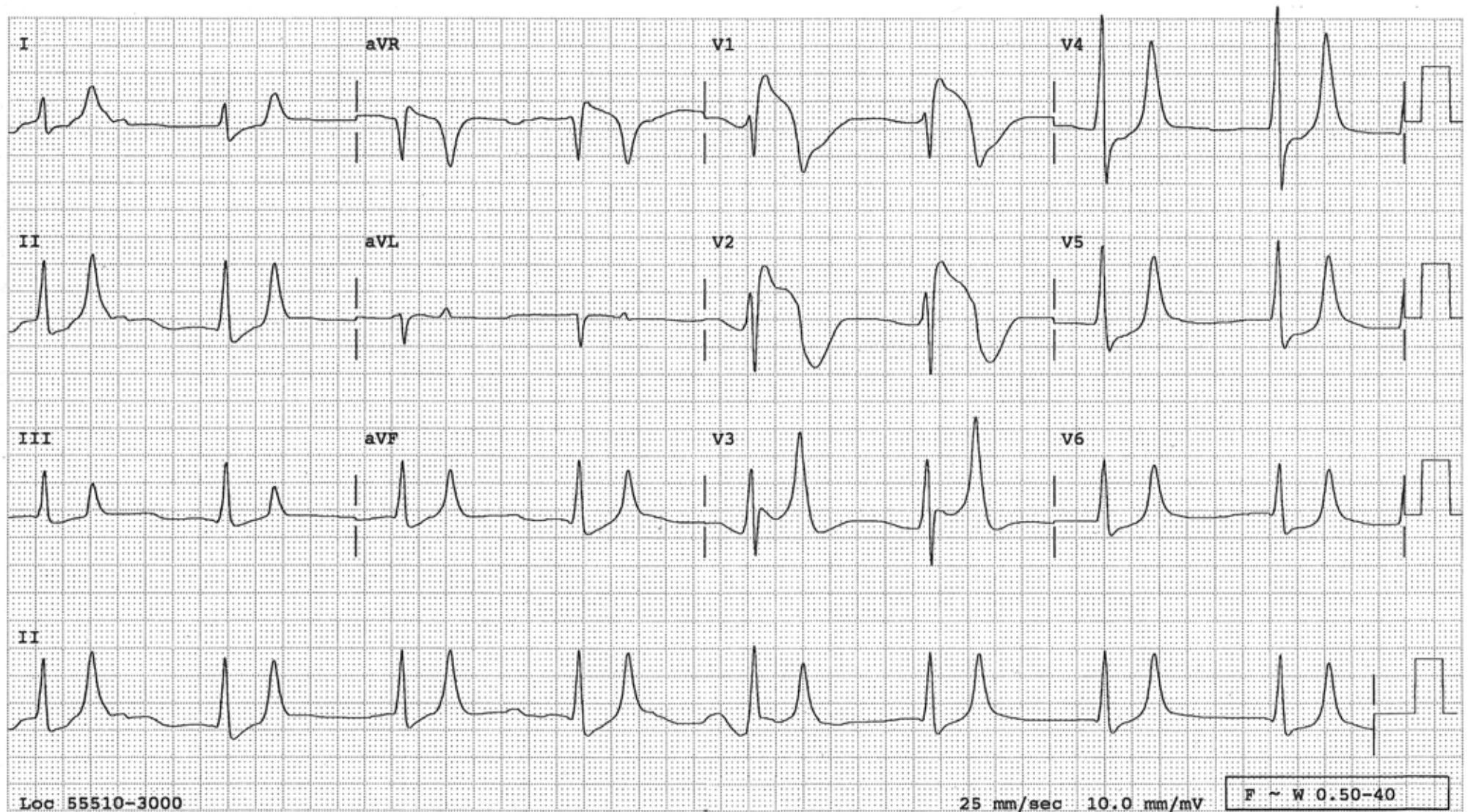
5-7 Sinus rhythm at a rate of about 85/minute. The P-R interval lengthens before every third P wave is blocked indicating Type I 2° AV block. This is not complete AV block since the R-R interval is not regular. In fact, the QRSs are paired.

Dx: Sinus rhythm with Type I 2° AV block (3:2 AV Wenckebach phenomenon)



5-8 A regular rhythm at a rate of 86/minute. QRSs are narrow. The negative deflection between the QRSs in V_1 is actually a P wave which can be appreciated also in V_3 . The P-R interval is prolonged to 350 milliseconds reflecting 1° AV block. The deflection after the first QRS in aVR, aVL and aVF is an artifact.

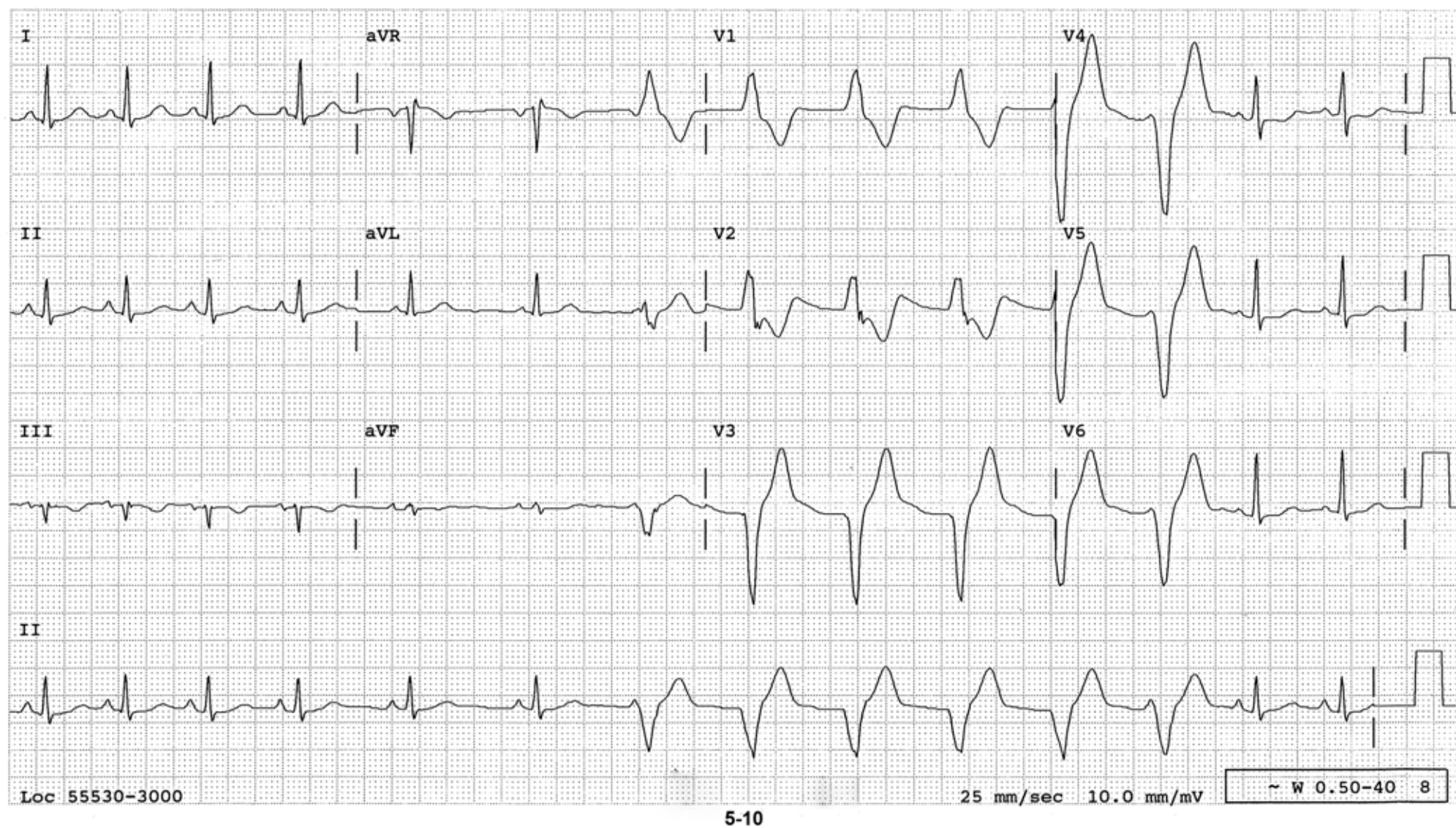
- Dx:
1. Sinus rhythm
 2. 1° AV block
 3. LAE



5-9

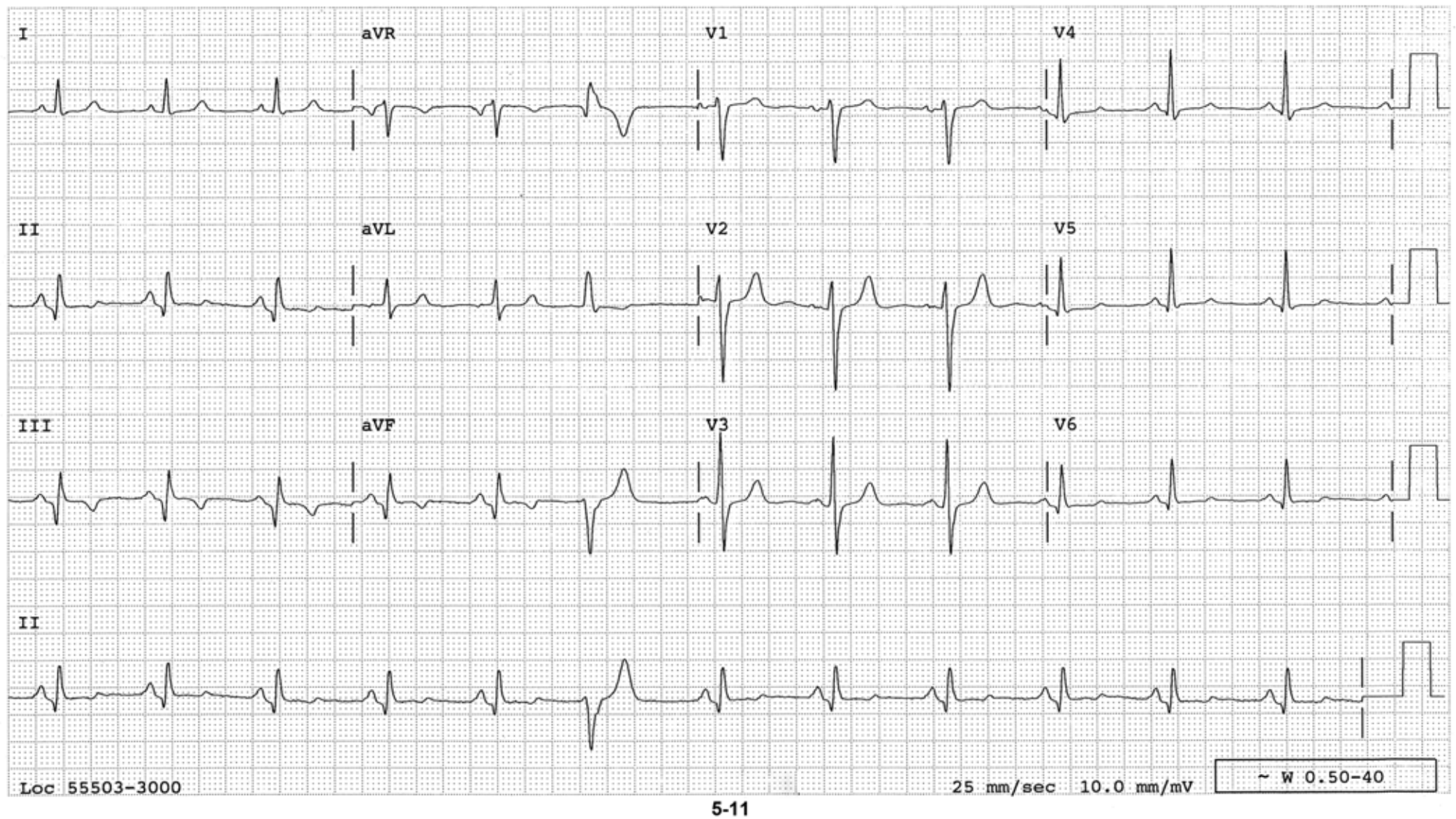
- 5-9 A regular rhythm at a rate of about 47/minute. QRSs are wide and no P waves can be recognized definitely. T waves are tall, narrow, tented and pointed. These are all features of hyperkalemia. ST elevation in V_1 and V_2 suggests acute infarct, but this is an example of a pseudoinfarction pattern (acquired Brugada ECG pattern) from hyperkalemia. Hyperkalemia is known to disable Na channel transiently causing this ECG pattern. What is the rhythm? No definite P waves are seen and the QRSs are wide, making some to think of ventricular rhythm, but it is not. In hyperkalemia, the P waves become flatter and flatter and eventually disappear; hyperkalemia made atrial muscle inexcitable. Still the impulse from the sinus node is conducted to the AV node through the specialized internodal tract(s), then to the ventricles, and this rhythm is called sinoventricular rhythm. This is the physiological proof for the presence of the internodal tract(s) even though it has not been convincingly identified histologically.

Dx: Hyperkalemia with sinoventricular rhythm and pseudoinfarction pattern (acquired Brugada ECG pattern)



- 5-10 Normal sinus rhythm which gradually slows down to yield to a regular, wide QRS rhythm (accelerated idioventricular rhythm) and then speeds up to regain control of the ventricles as can be seen in the last two complexes. A P wave merging into the QRS is seen in the first complex of the wide QRS rhythm, and a P wave emerging from the QRS can be seen with the last complex of the wide QRS rhythm. In between, the P waves are buried within the QRSs. This accelerated idioventricular rhythm is seen mostly during acute MI, and is often a sign of reperfusion.

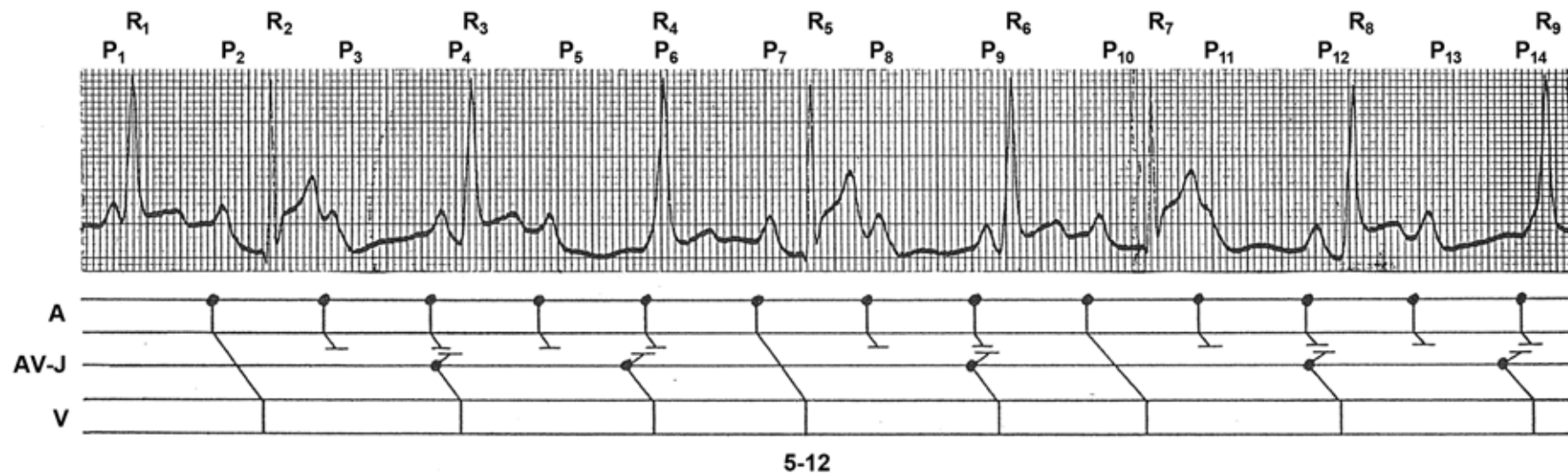
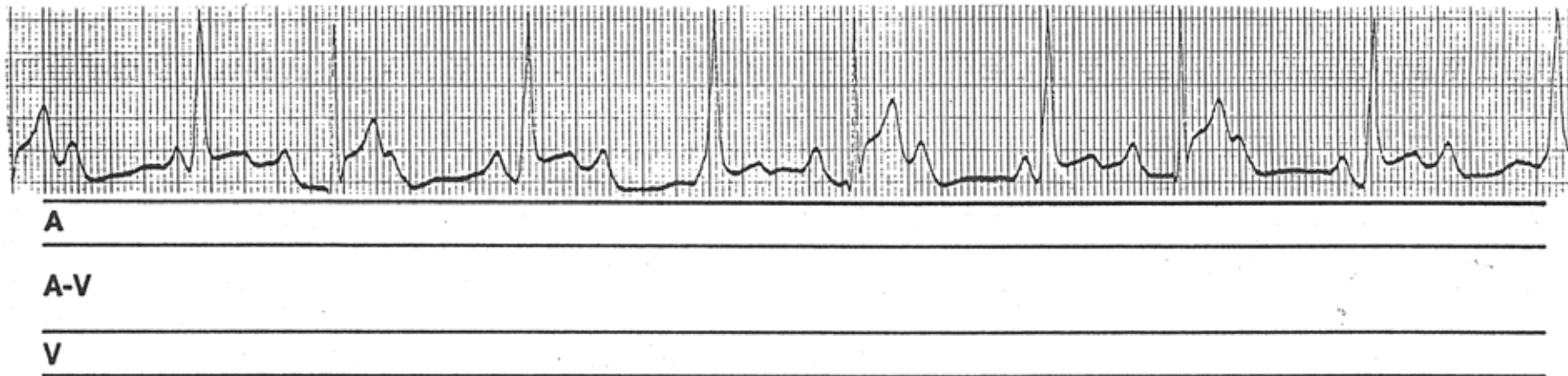
Dx: NSR with a short run of accelerated idioventricular rhythm



5-11 Normal sinus rhythm at a rate of 74/minute. Q waves with a terminal T wave inversion in the inferior leads are good for recent inferior MI. One ventricular premature complex is present.

- Dx:
1. NSR
 2. Inferior infarct, recent
 3. PVC

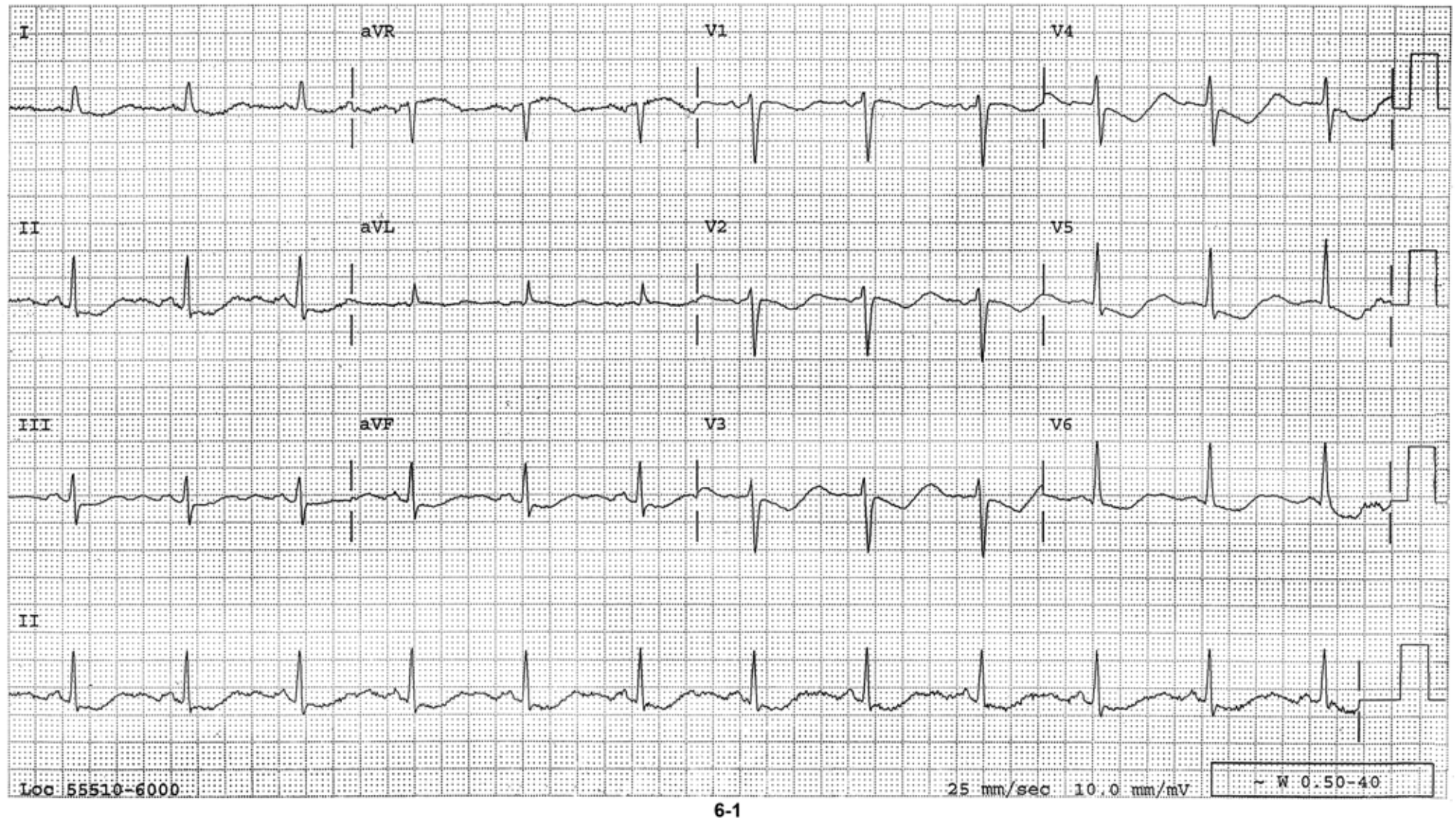
Please draw the ladder diagram in the space provided below.



5-12

There are more P waves than R waves and the P waves do not seem to have any fixed relationship to the R waves, raising a possibility of complete AV block. However, the fact that the R waves not occurring regularly is a strong evidence against complete AV block (in complete AV block, escape rhythm is very regular). The only explanation for R₂, R₅, and R₇ to occur with a shorter R-R interval than others is that they are induced by the P wave (capture beats). These capture beats also contain additional important information, i.e. AV conduction time (P-R interval) in this patient (e.g. P₂R₂, P₇R₅, and P₁₀R₇). Any P-R interval shorter than this (e.g. P₁R₁, P₄R₃, P₉R₆, P₁₂R₈) means that the R wave is not induced by the P wave. Armed with this information, one can start the ladder diagram from R₂. P₂ is conducted to R₂. P₃ is blocked. P₄ is going to be conducted to the ventricles, but two P-P intervals (P₂-P₄) are longer than the junctional escape interval and a junctional escape beats and AV dissociation results. P₅ is blocked. P₆ and R₄ (junctional escape beats) are again dissociated. The primary disorder in this tracing is 2:1 AV block. Junctional escape beats and AV dissociation are all obligatory secondary responses to this primary disorder. This tracing is from a patient with an acute inferior MI.

SECTION 6



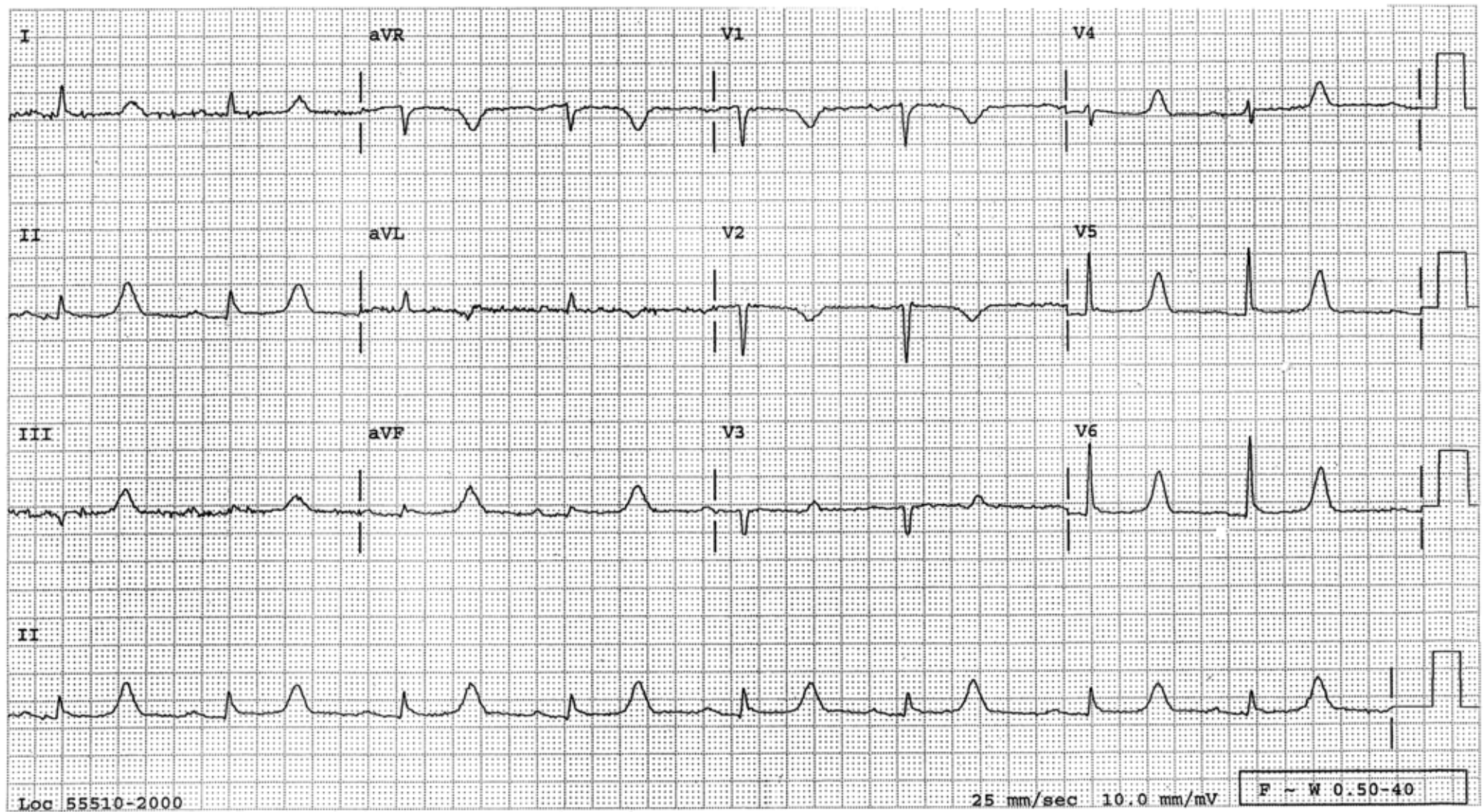
6-1 Mildly inverted T waves followed by prominent U waves with a prolonged Q-T-U interval are characteristic of hypokalemia.

Dx: Hypokalemia



6-2 Wide QRS complexes, diminished P wave voltage, a prolonged P-R interval and tall, narrow, tented and pointed T waves are all characteristic of hyperkalemia.

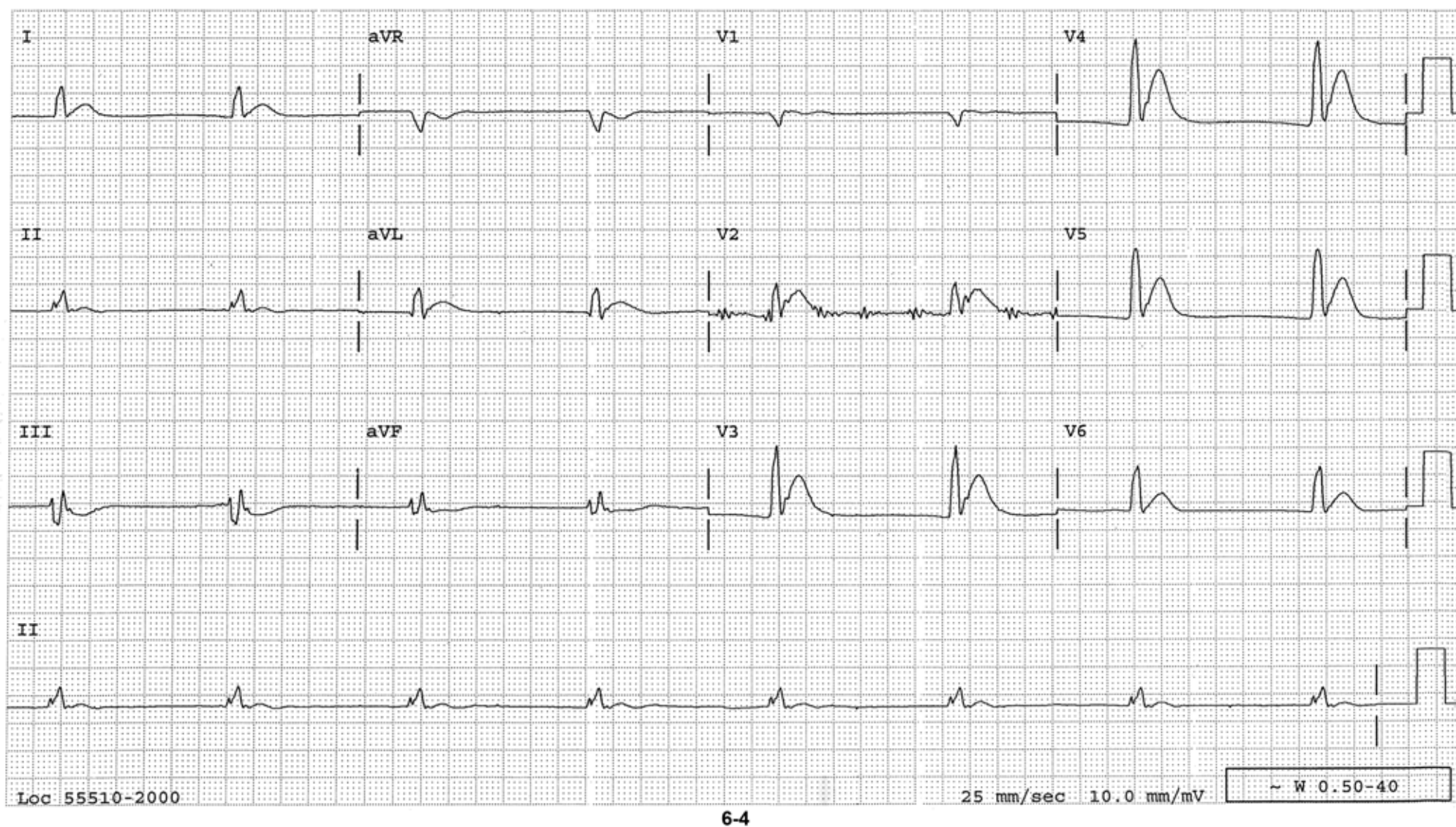
Dx: Hyperkalemia



6-3

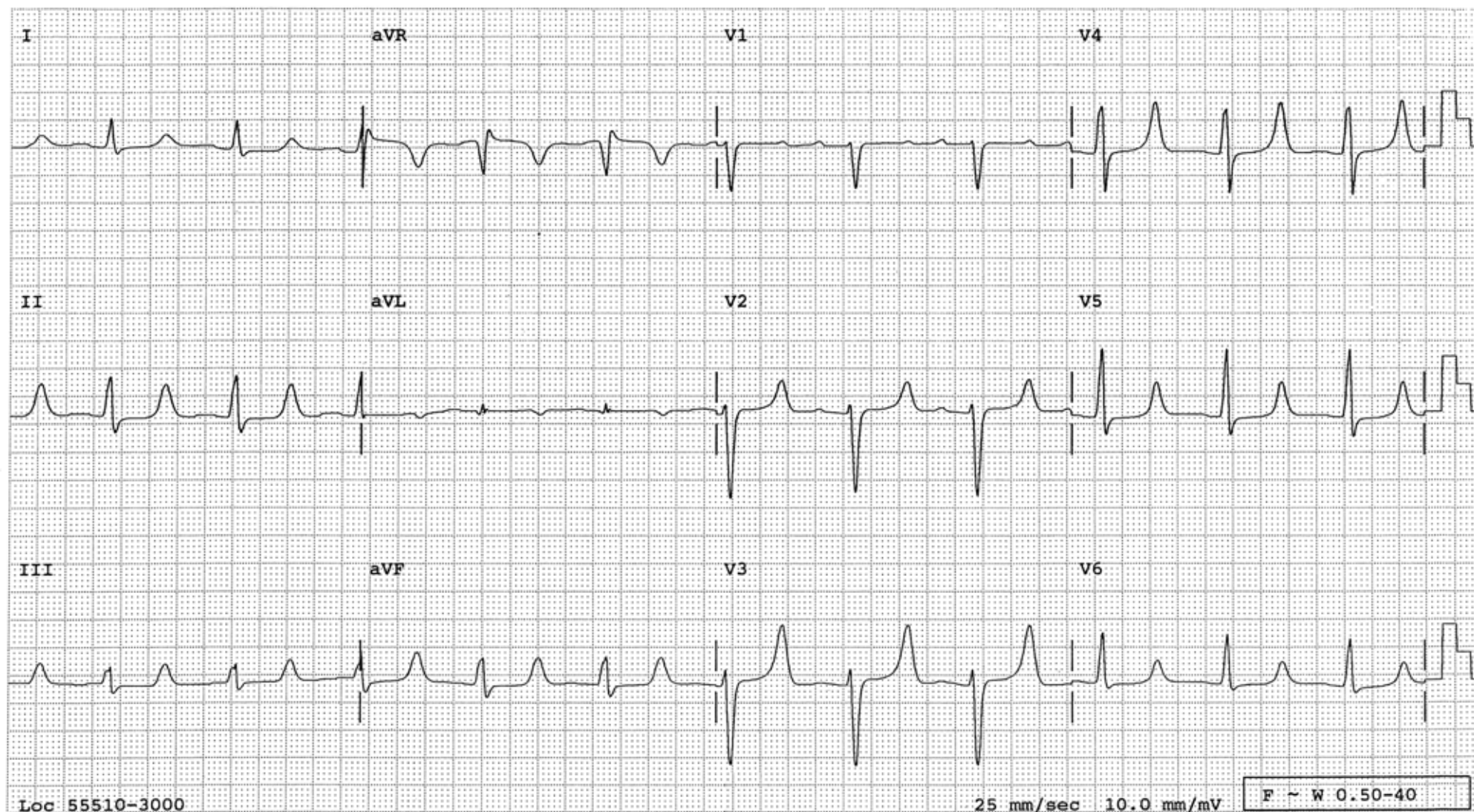
6-3 A long Q-T interval made of a long ST-segment with a delayed onset of the T wave is characteristic of hypocalcemia.

Dx: Hypocalcemia



6-4 A short Q-T interval without hardly any ST-segment is characteristic of hypercalcemia. Other findings not related to hypercalcemia are IVCD and junctional escape rhythm.

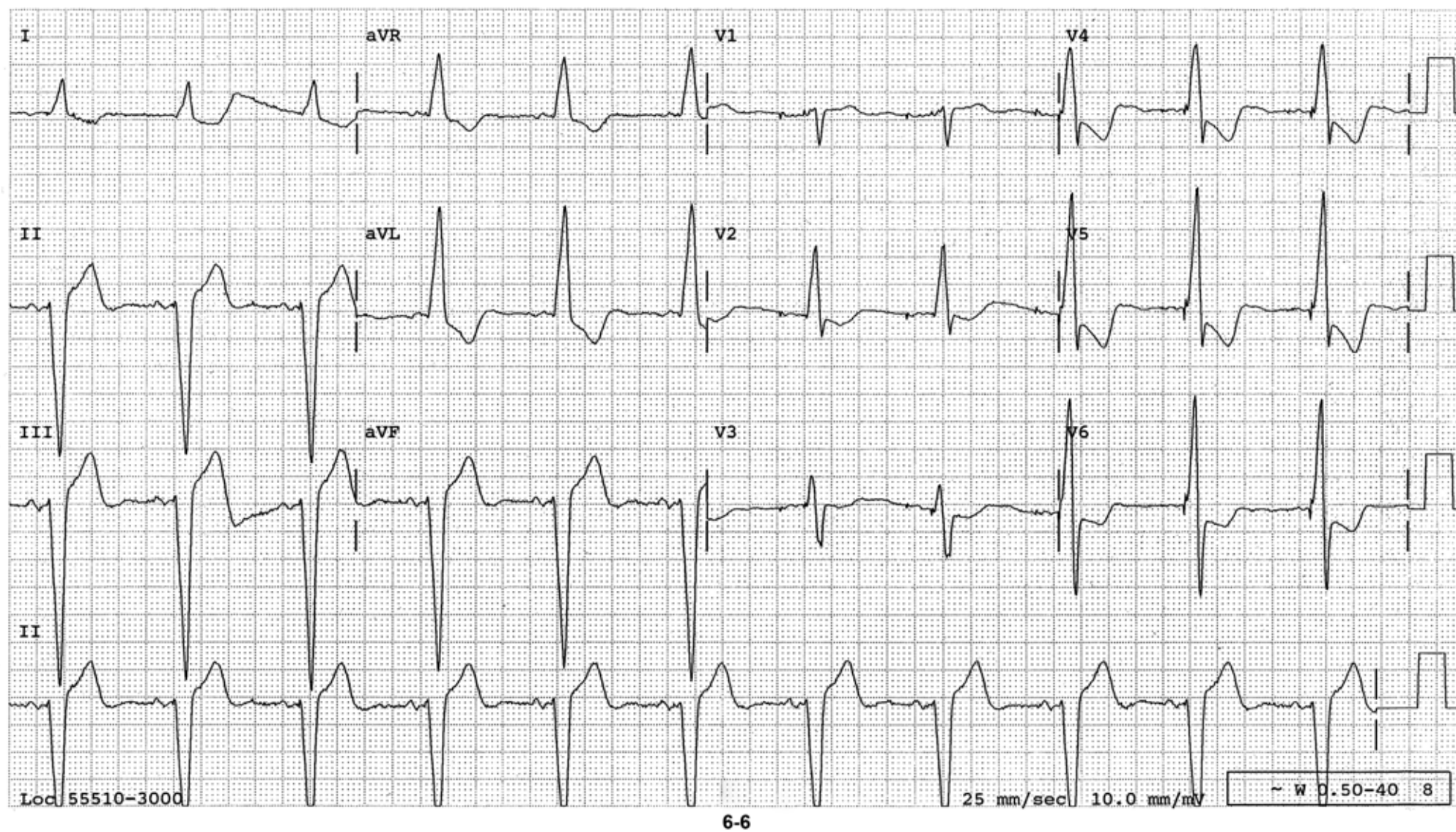
Dx: Hypercalcemia



6-5

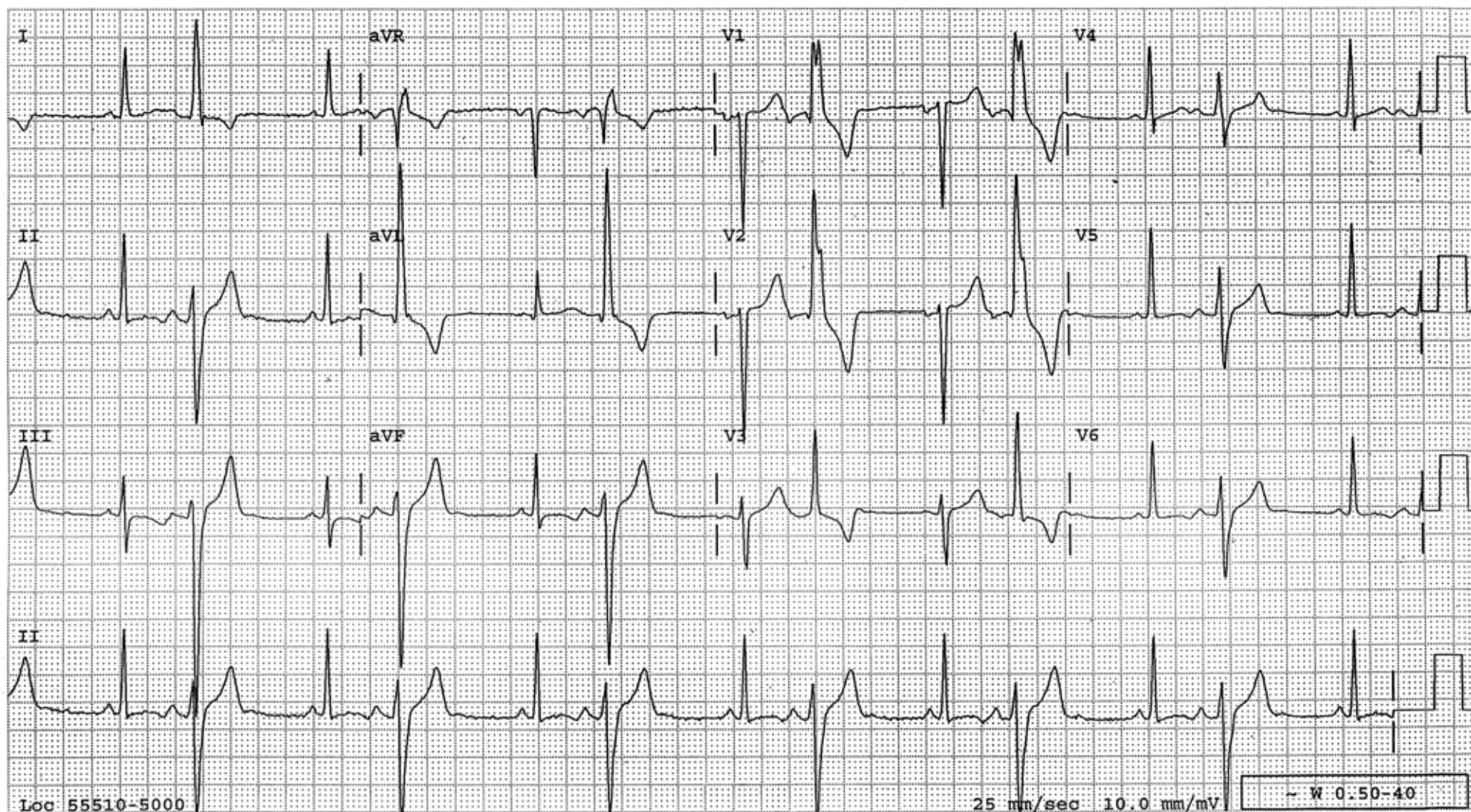
6-5 The Q-T interval is prolonged which is due to a prolonged ST-segment with delayed onset of T waves. These findings are characteristic of hypocalcemia. Additionally, the T waves are tall, narrow and pointed, indicating hyperkalemia as well. This combination of electrolyte problems is typically seen in renal failure.

Dx: Hyperkalemia and hypocalcemia



6-6 Lead V₁ shows pacemaker spikes, one for the atrium and the other for the ventricle.

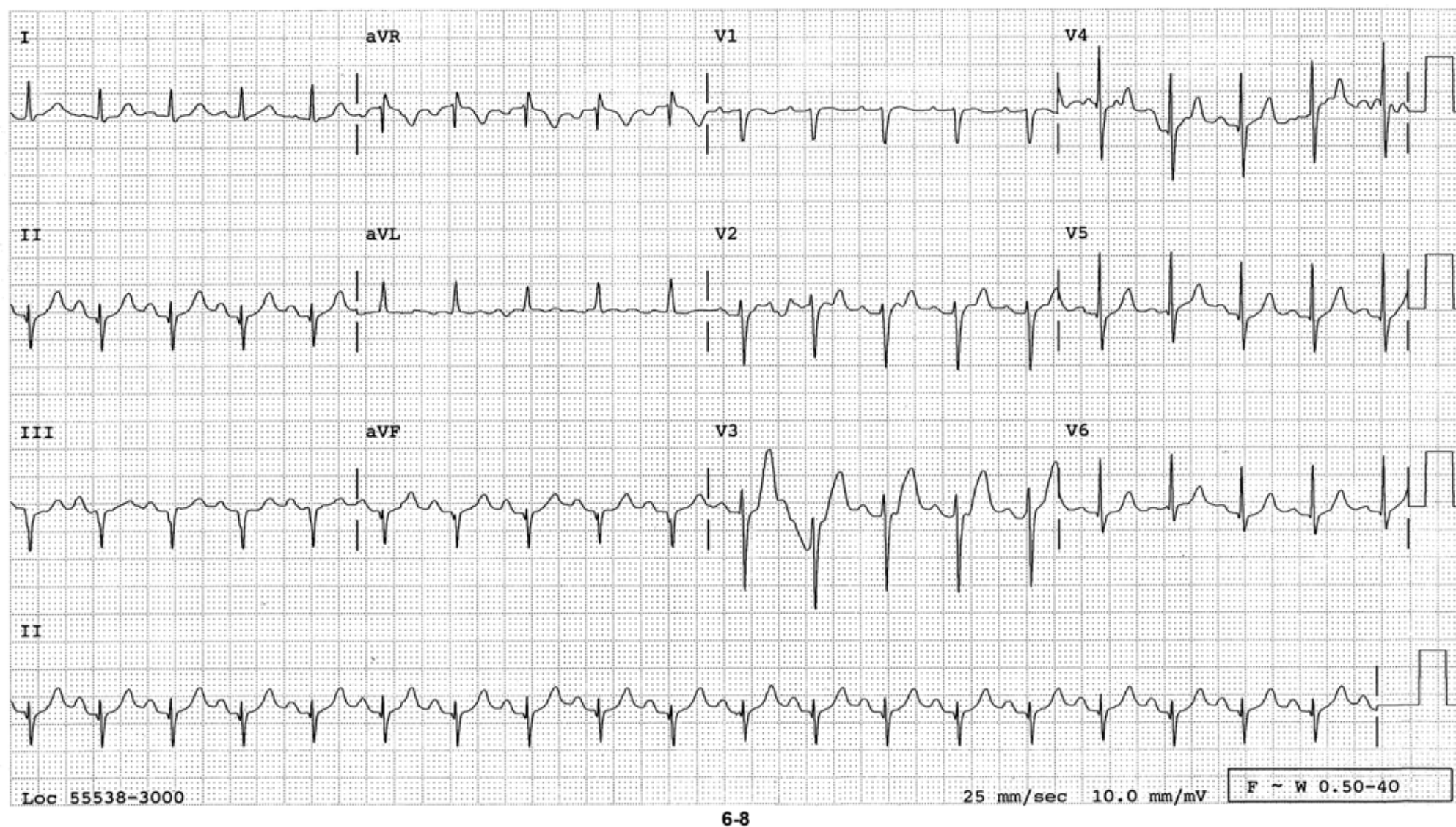
Dx: AV sequential paced rhythm



6-7

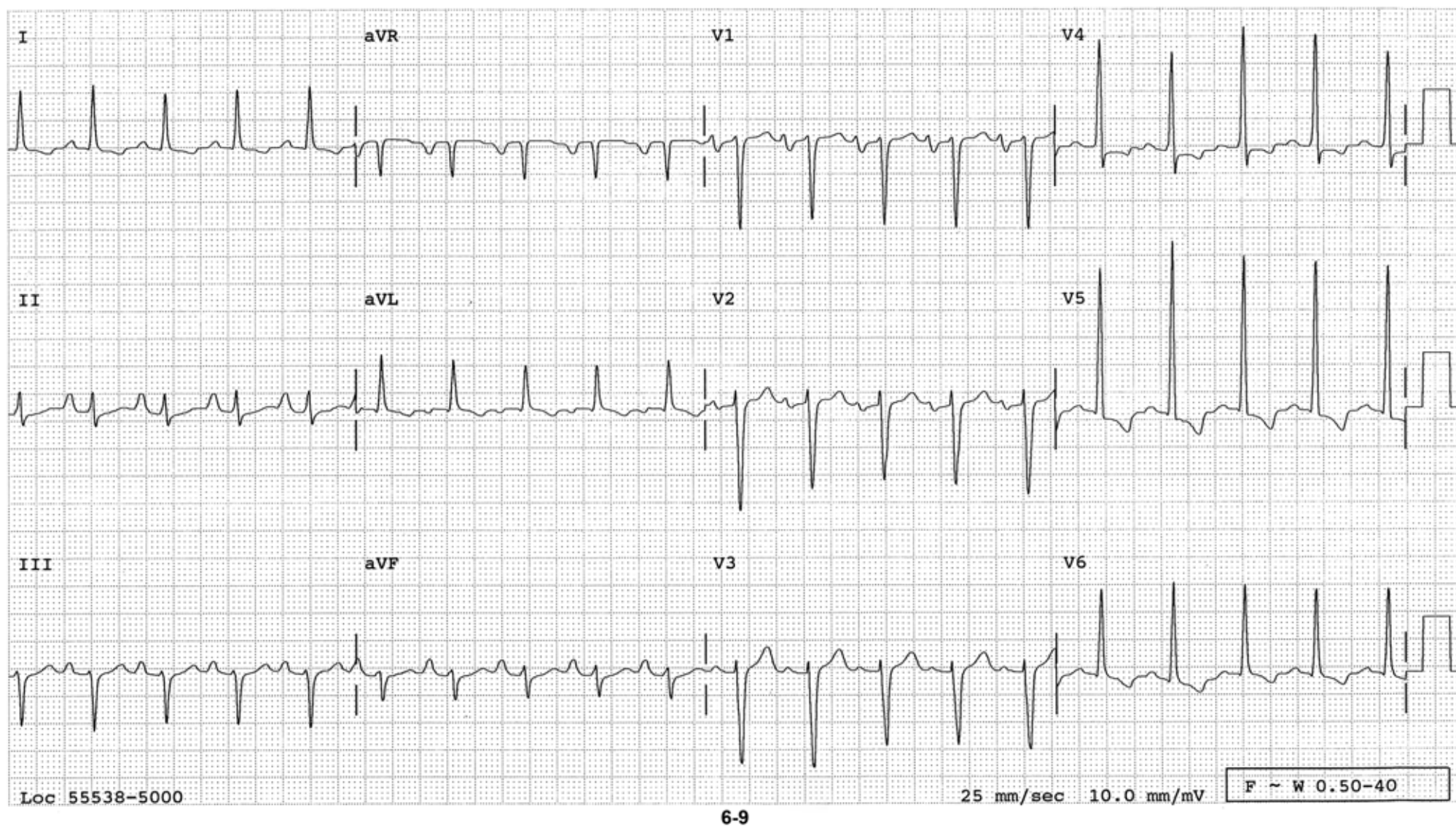
- 6-7 Every other beat appears to be a ventricular premature beat (ventricular bigeminy). However, there is a P wave in front of these premature beats, best seen in V_1 - V_2 , and these are atrial premature beats conducted aberrantly. Aberrant conduction occurs simply because two bundle branches have different lengths of refractory period. So if an impulse occurs at a time when one bundle branch is still refractory while the other BB has recovered, the impulse will conduct through whichever has recovered, bypassing the other BB, resulting in an aberrant conduction. Usually it's the RBB which has the longer refractory period, which is the reason why aberrant conduction more often manifests as RBBB type, even though it may be reversed in a given patient at different times or in different patients.

Dx: Atrial bigeminy with aberrant conduction



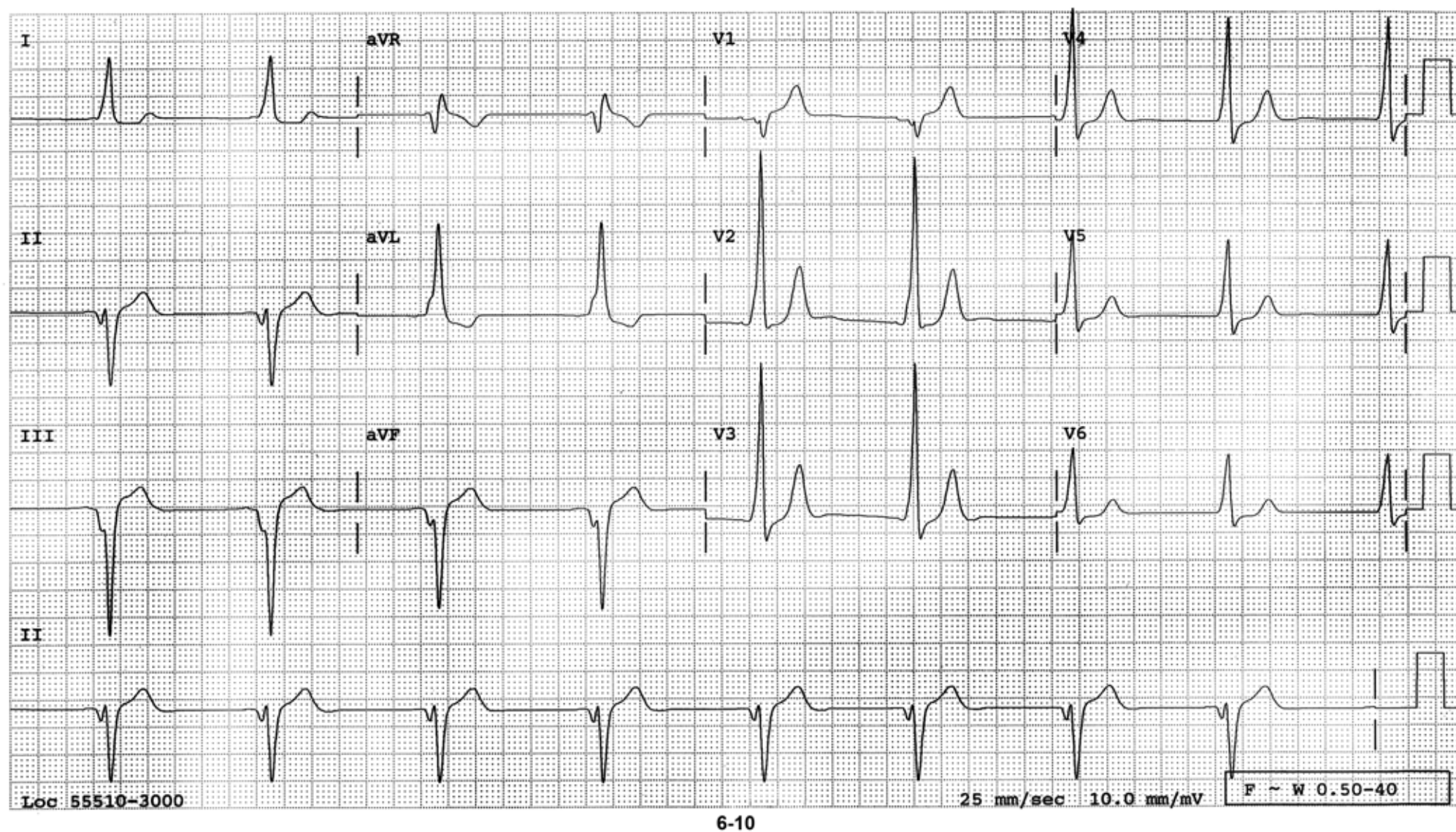
6-8 Sinus tachycardia at a rate of 115/minute. The mean QRS axis is deviated to the left indicating LAFB. QS pattern in leads III and aVF may be due to LAFB alone or that combined with old inferior MI. The lead II holds the key. If it begins with a Q wave no matter how small it is, old inferior MI can be called. If it begins with an R wave, it is just LAFB. The lead II in this case begins with a small Q wave and old inferior MI can be diagnosed.

- Dx:
1. Sinus tachycardia
 2. LAFB
 3. Old inferior infarct



6-9 Sinus tachycardia at a rate of 115/minute. P wave is taller than 2.5 mm in lead II indicating RAE. The P wave in V_1 is biphasic with a slurred transition from positive to negative deflection indicating LAE as well. Voltage criteria and ST-T changes for LVH are present.

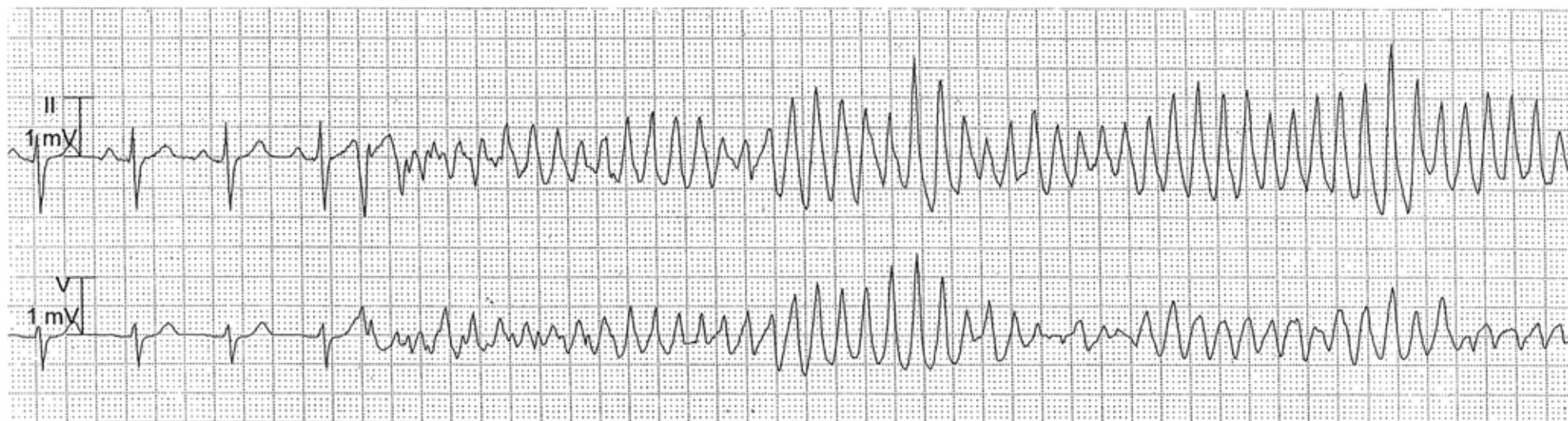
- Dx:*
1. Sinus tachycardia
 2. Biatrial enlargement
 3. LVH



6-10 A regular rhythm at a rate of 51/minute. The P waves are not readily recognizable and the QRSs are wide raising a possibility of ventricular rhythm. However, in leads I, III, aVF and V_2 the P waves are barely recognizable with a very short P-R interval. The upstroke of the QRS is slurred especially in leads I, aVL, V_2 and V_3 . These findings are diagnostic of WPW syndrome. The delta waves are negative in the inferior leads simulating Q waves of an infarct.

Dx: 1. NSR

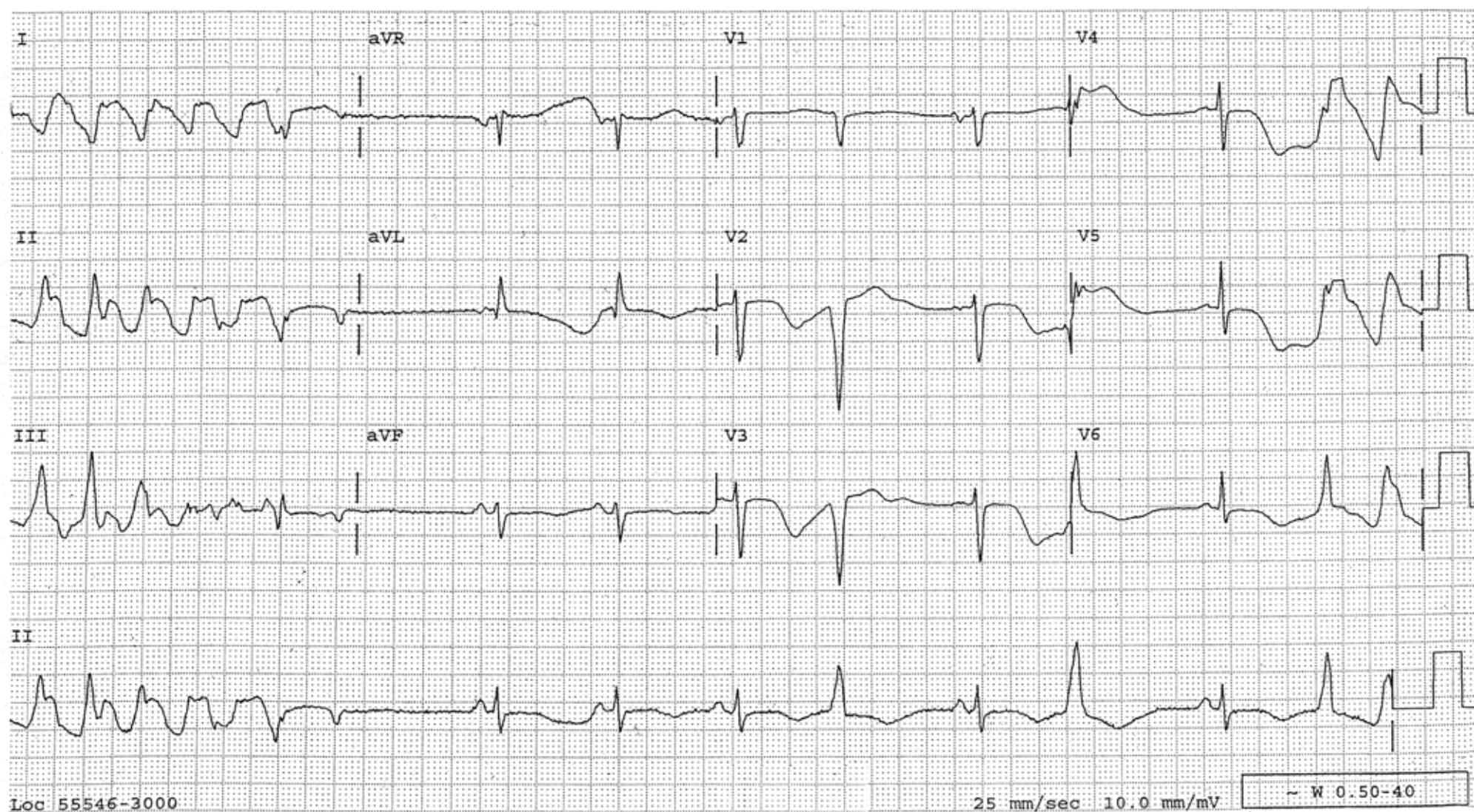
2. WPW syndrome simulating either inferior infarct or idioventricular rhythm



6-11

- 6-11 Normal sinus rhythm is present initially, which is followed by polymorphic VT. If the Q-T interval is long with the sinus beats, that polymorphic VT will be called *Torsade de Pointes*, which is not the case here. It is an important distinction because the causes and treatments are different in these two conditions. Polymorphic VT is more often 2° to myocardial ischemia or ventricular dysfunction and electrical cardioversion is the treatment of choice if the rhythm is sustained, or amiodarone for recurrent short runs. *Torsade de Pointes* is 2° to long Q-T interval and also has to be electrically converted if sustained, but MgSO₄ IV bolus is the treatment of choice for recurrent episodes, while withholding or avoiding the QT prolonging drugs.

Dx: Sinus rhythm going into polymorphic ventricular tachycardia

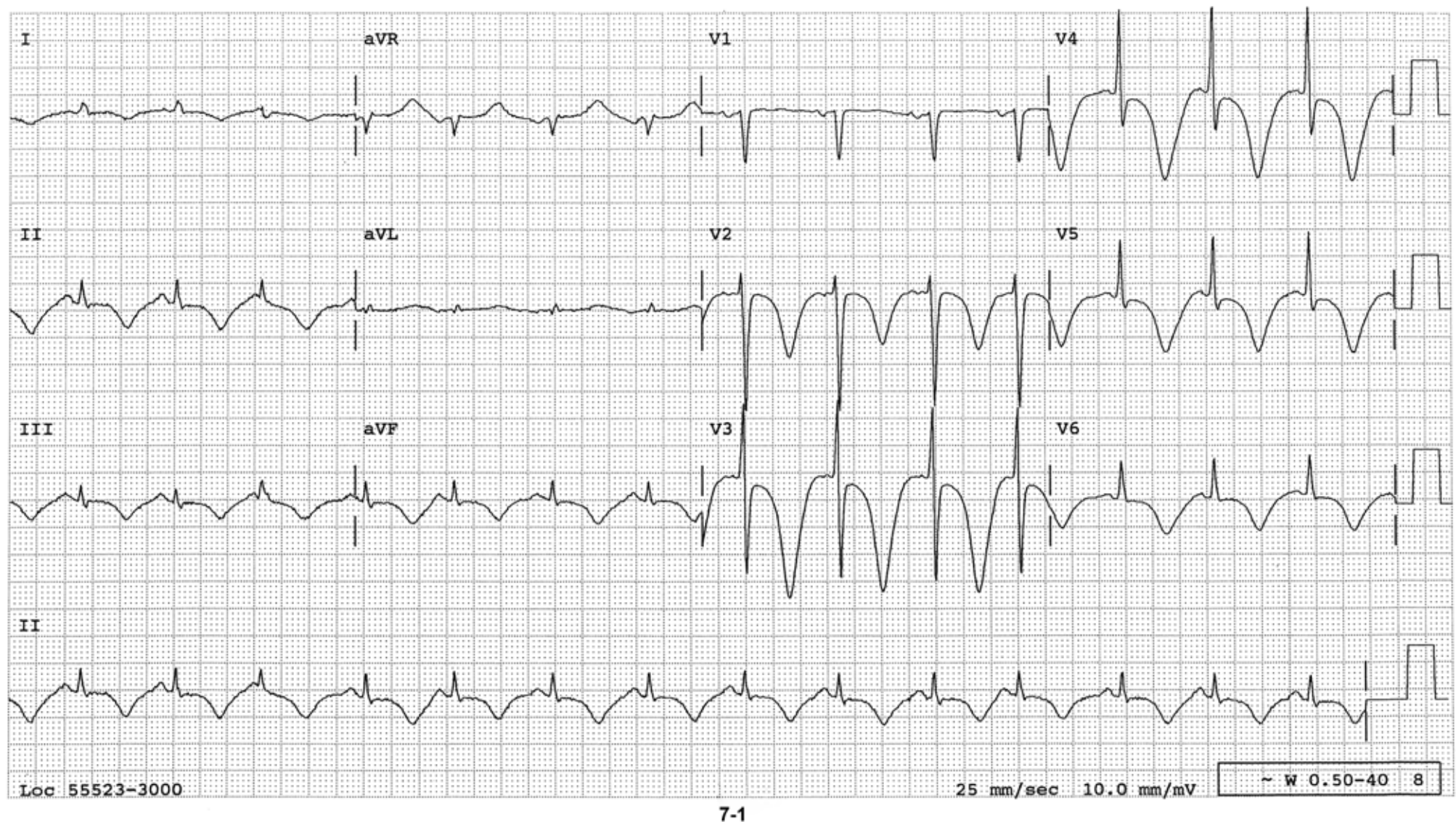


6-12

6-12 Polymorphic VT is present at the beginning of the tracing. During sinus rhythm the Q-T interval is very long and this polymorphic VT will be specifically called *Torsade de Pointes*. *Torsade de Pointes* is 2° to long Q-T interval and can be electrically converted if sustained. MgSO₄ IV bolus is the treatment of choice for recurrent episodes, while withholding or avoiding QT prolonging drugs.

Dx: *Torsade de Pointes*

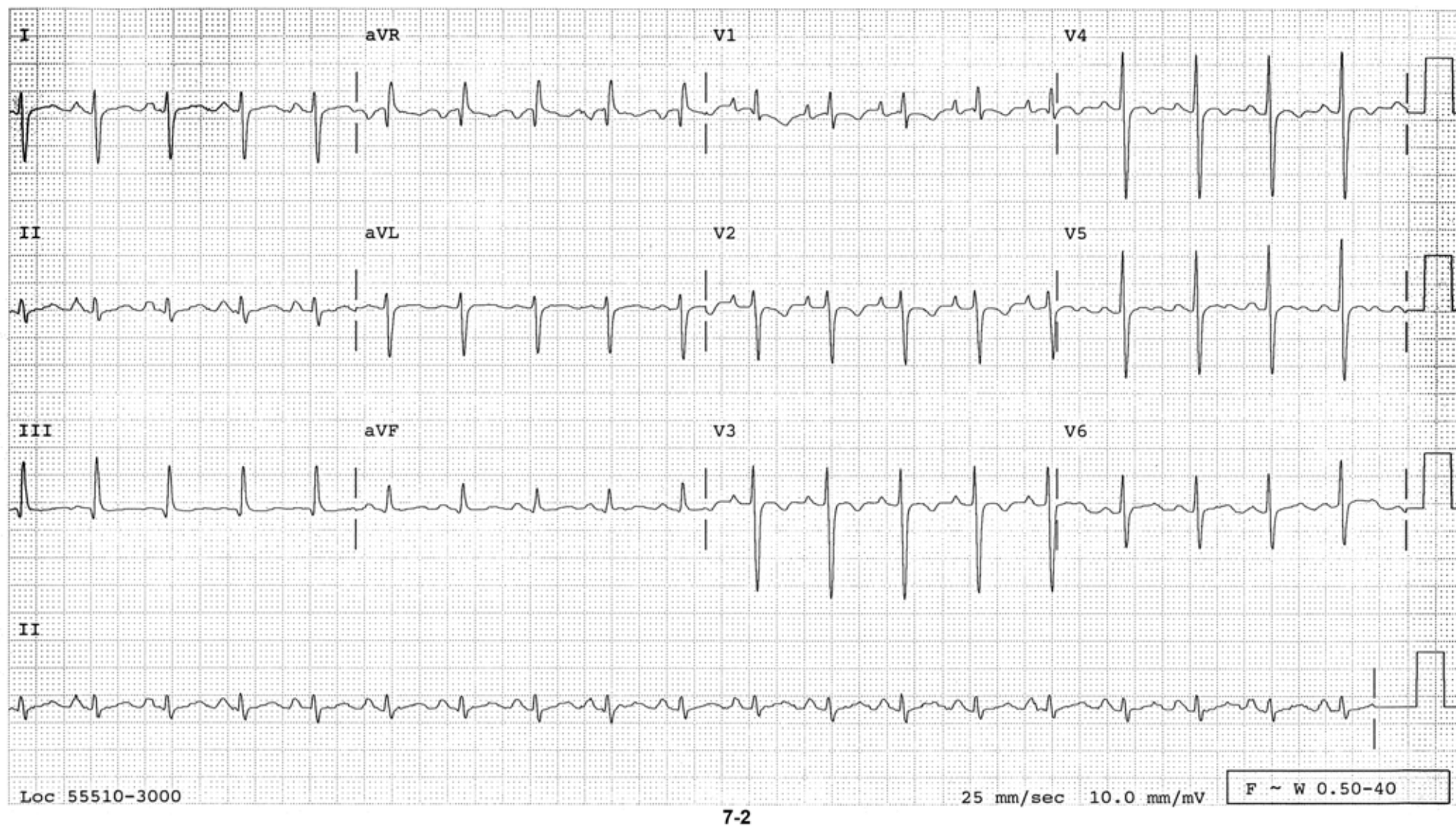
SECTION 7



7-1 Normal sinus rhythm at ~85/minute. T waves are deeply and symmetrically inverted with a long Q-T interval. These findings are often seen in acute CNS events, such as subarachnoid hemorrhage or head trauma, hence used to be called neurogenic T wave changes. However, these ECG findings are not unique to CNS events since the same findings have been observed in a variety of other acute medical conditions, including emotional stress, more often in elderly women (stress cardiomyopathy). At the onset, many patients develop mild ST-segment elevation indistinguishable from that of acute infarction. On echocardiogram or ventriculogram the distal half of left ventricle is ballooned out (apical ballooning syndrome or Takotsubo cardiomyopathy) with a complete recovery. The mechanism is not well-understood, but catecholamine surge seems to be the common denominator of this entity.

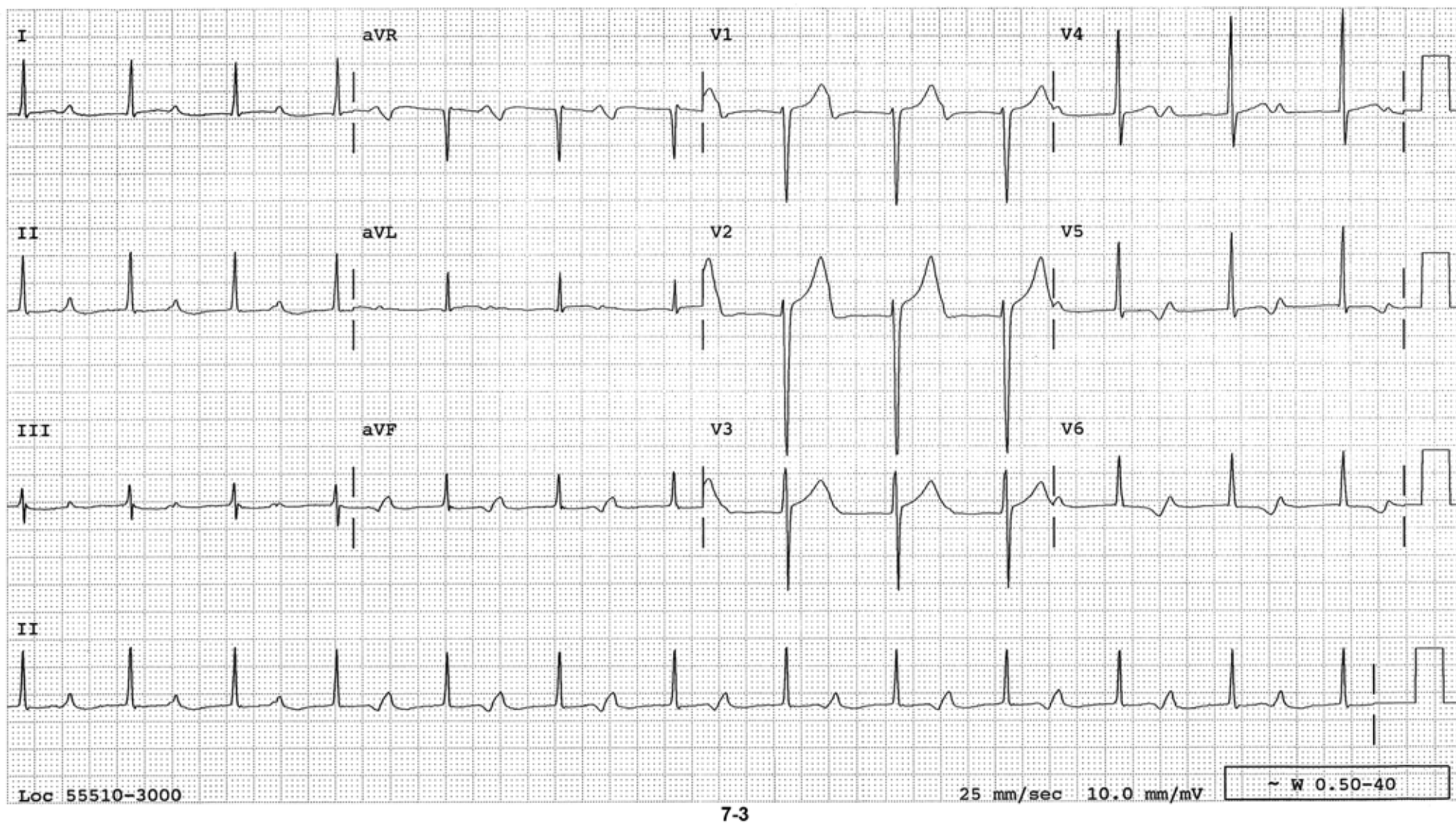
Dx: 1. NSR

2. T wave changes suggestive of stress cardiomyopathy



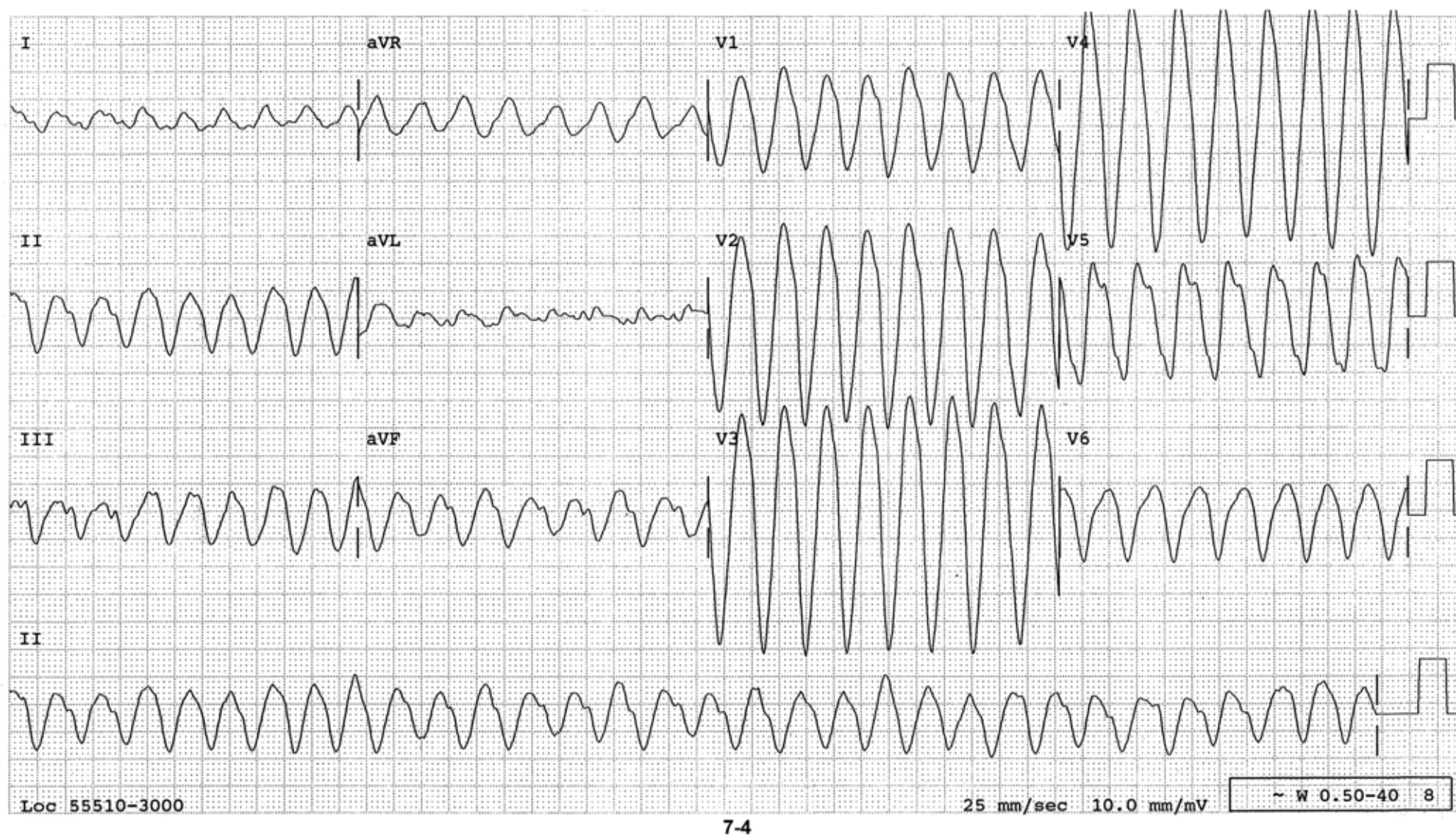
7-2 Sinus tachycardia at a rate of 112/minute. Mean QRS axis is shifted markedly to the right. R wave is taller than the S wave in V_1 . T waves are inverted predominantly in the right precordial leads. There is a deep S wave in V_6 . These are all diagnostic features of RVH.

- Dx: 1. Sinus tachycardia
2. RVH



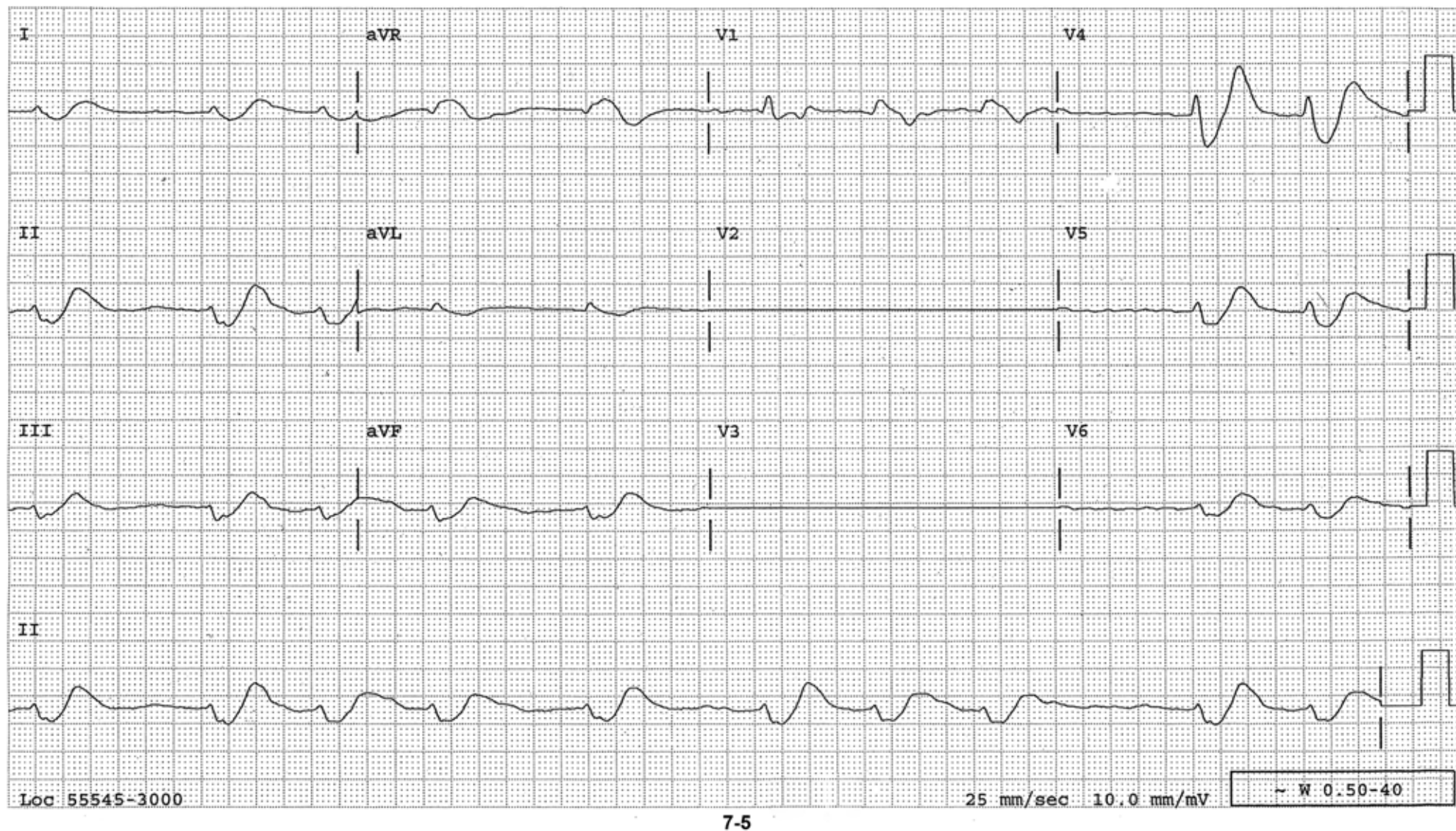
7-3 A regular rhythm at a rate of 75/minute. QRSs are narrow. P waves are not readily recognizable at first glance, but they are at the end of the T waves which is better appreciated in V_4 or V_1 . This is an example of 1° AV block with a P-R interval of about 480 milliseconds. Voltage criteria and ST-T changes for LVH are present.

- Dx*
1. Sinus rhythm with 1° AV block
 2. LVH



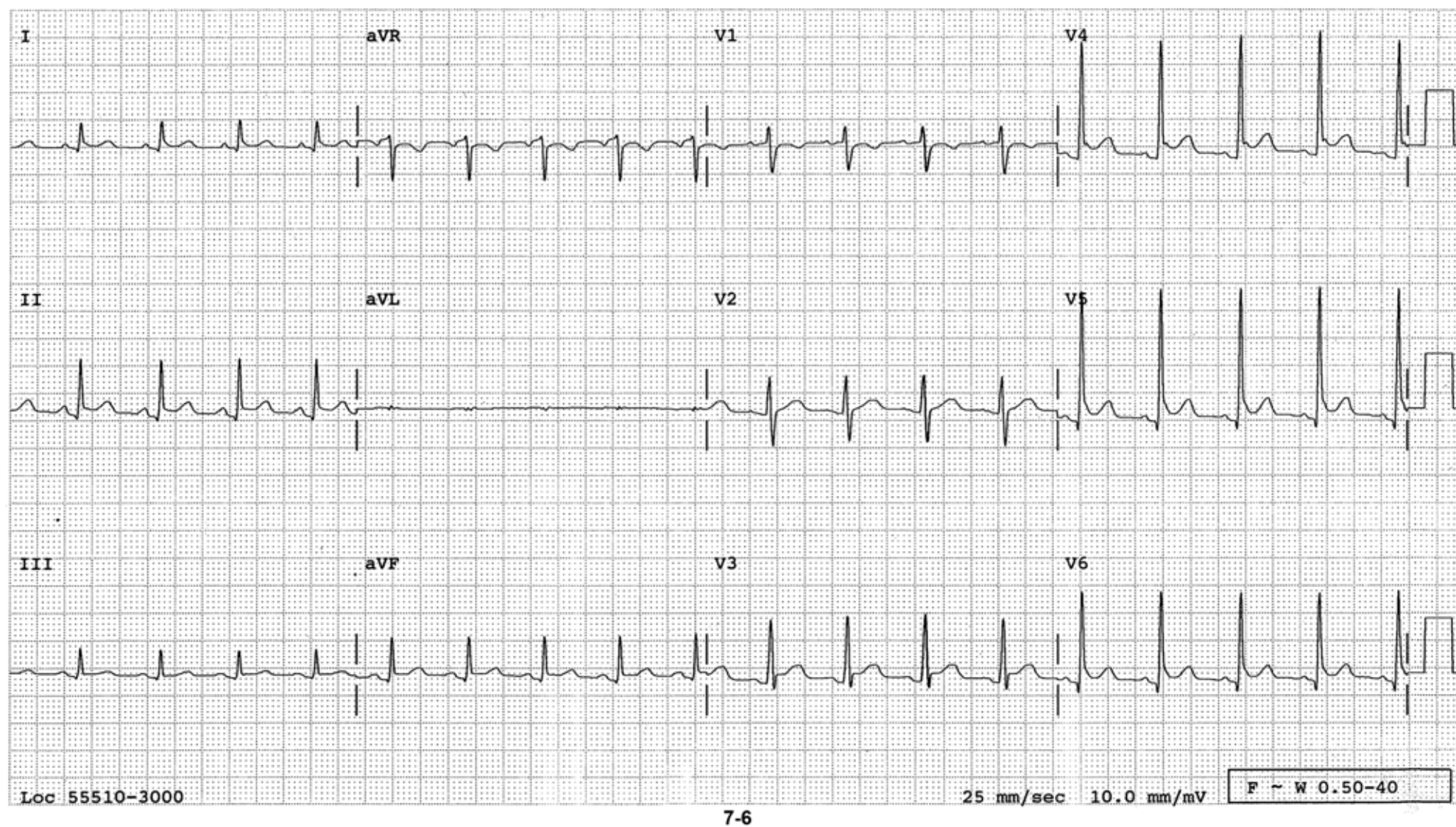
- 7-4 A regular wide-QRS tachycardia is present. When the rate is this fast, use two R-R intervals rather than one R-R interval to estimate the heart rate (HR), then double it. The two R-R intervals are three big boxes wide, which will make the HR of 100/minute. Therefore, the actual HR is 200/minute (100 x 2). It is difficult to tell where the QRS begins and where it ends. At any rate, the QRS is wider than 200 milliseconds, and the rhythm is VT. In SVT with aberrant conduction, the QRS width seldom exceeds 140 milliseconds.

Dx: VT



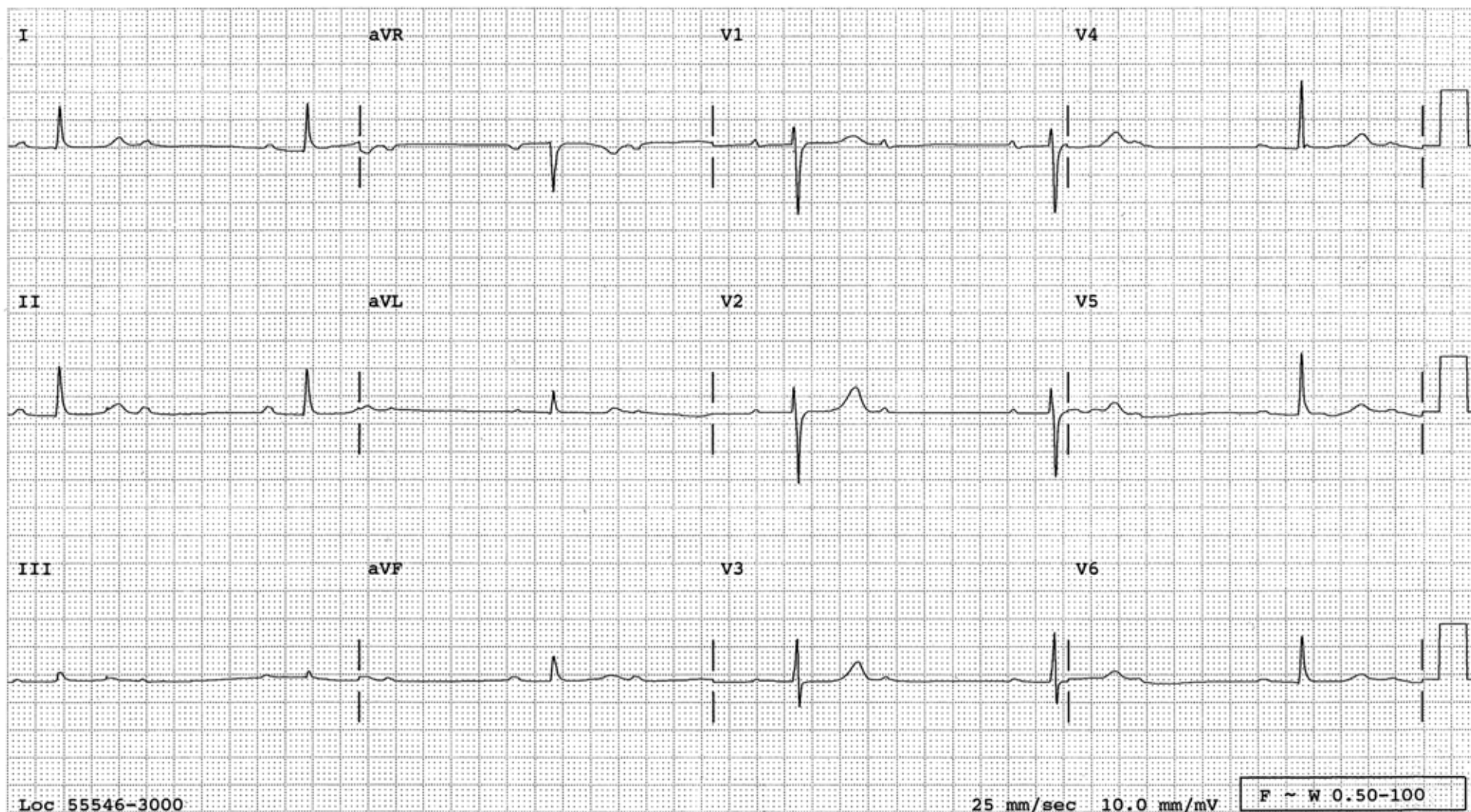
7-5 Irregularly irregular rhythm with no P waves indicates atrial fibrillation. QRSs are wide, merging into prominent T waves especially in V_4 and the tracing is highly suggestive of hyperkalemia. The serum K^+ was 7.8 mEq/L. Straight lines in V_2 and V_3 indicate that the electrodes are not placed rather than pauses or asystole. Pauses will not result in a straight line as if someone drew a line with a ruler, as in this case.

- Dx:*
1. Atrial fibrillation
 2. Findings highly suggestive of hyperkalemia



7-6 Sinus tachycardia at a rate of 107/minute. QRS voltage is increased in the precordial leads. ST-segment is elevated diffusely and PR-segment is depressed; findings good for pericarditis.

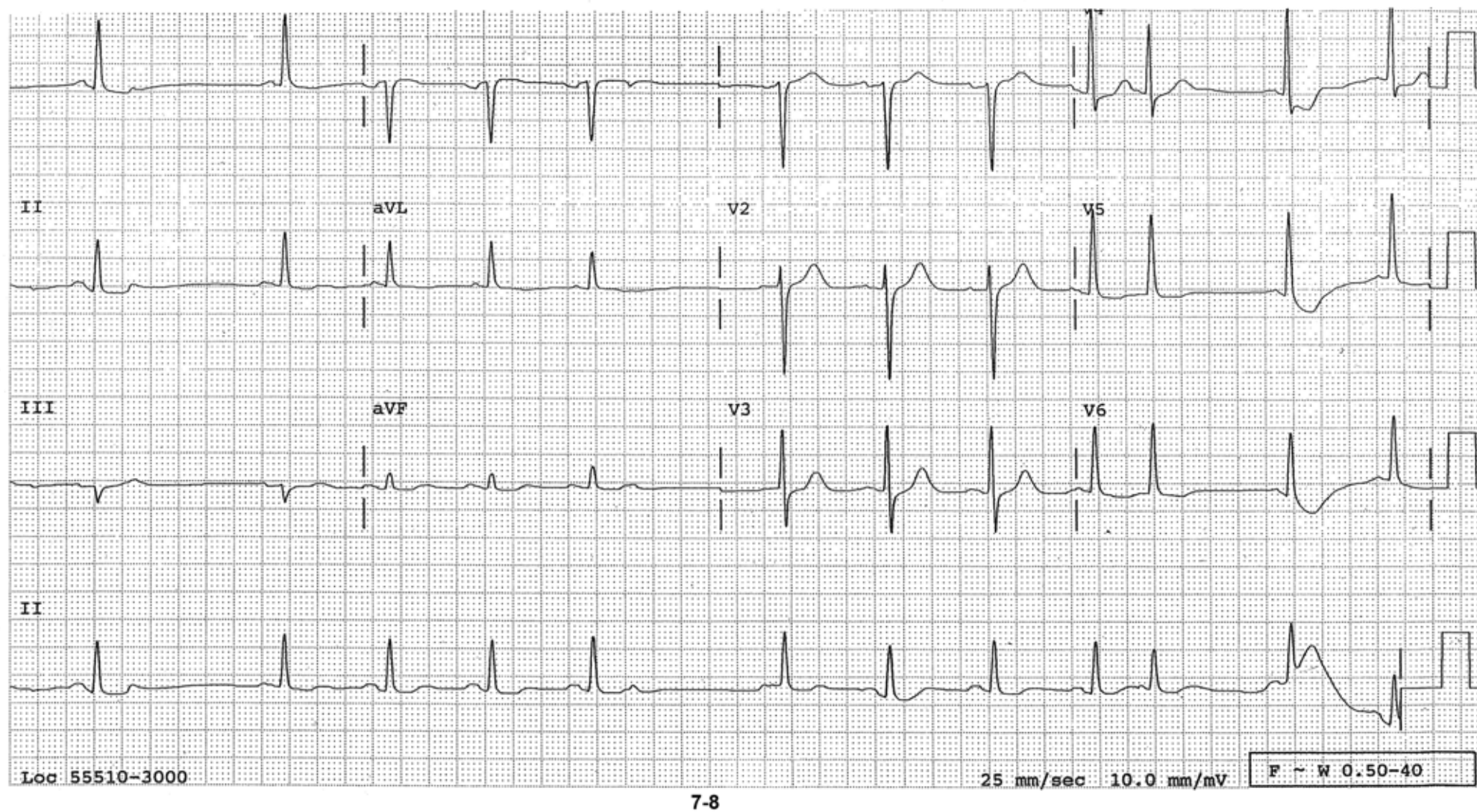
- Dx:
1. Sinus tachycardia
 2. LVH voltage
 3. Widespread ST elevation from pericarditis



7-7

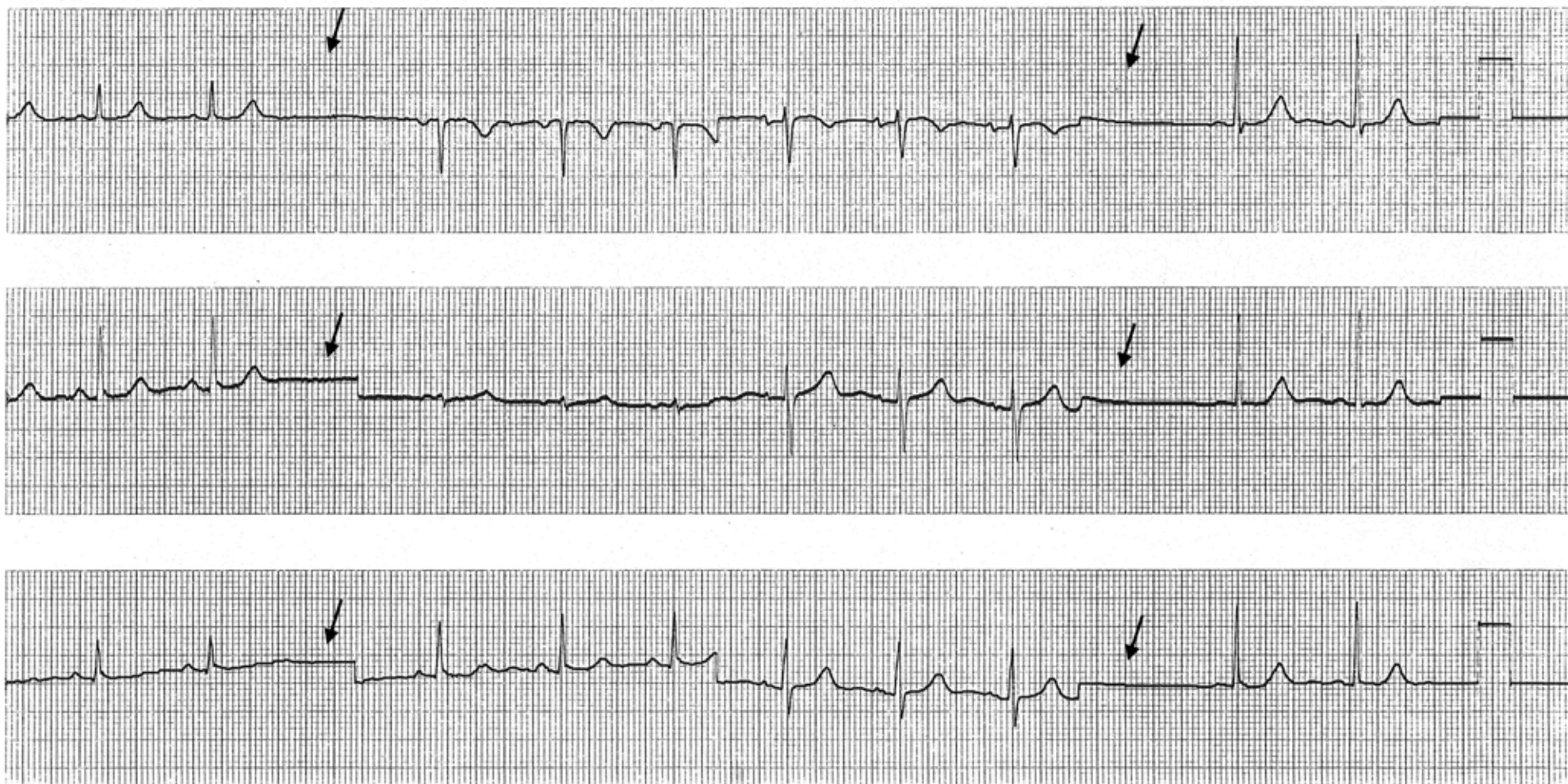
7-7 A slow and regular rhythm at a rate of 34/minute. QRSs are narrow. There are two P waves between the QRSs and they occur regularly. One of them has a fixed relationship to the following QRS indicating 2:1 AV block. Since the QRS is narrow, one would consider the block to be located within the AV node rather than below.

Dx: Sinus rhythm with 2:1 AV block, most likely block within the AV node.



7-8 The rhythm is mostly regular with occasional pauses. Careful examination during the pauses reveals a premature P wave superimposed on the T wave. At times, this premature P wave is conducted to the ventricles (the third complex from the end) supporting that these are indeed nonconducted PACs. An artifact distorts the ST-segment of the second from the last QRS.

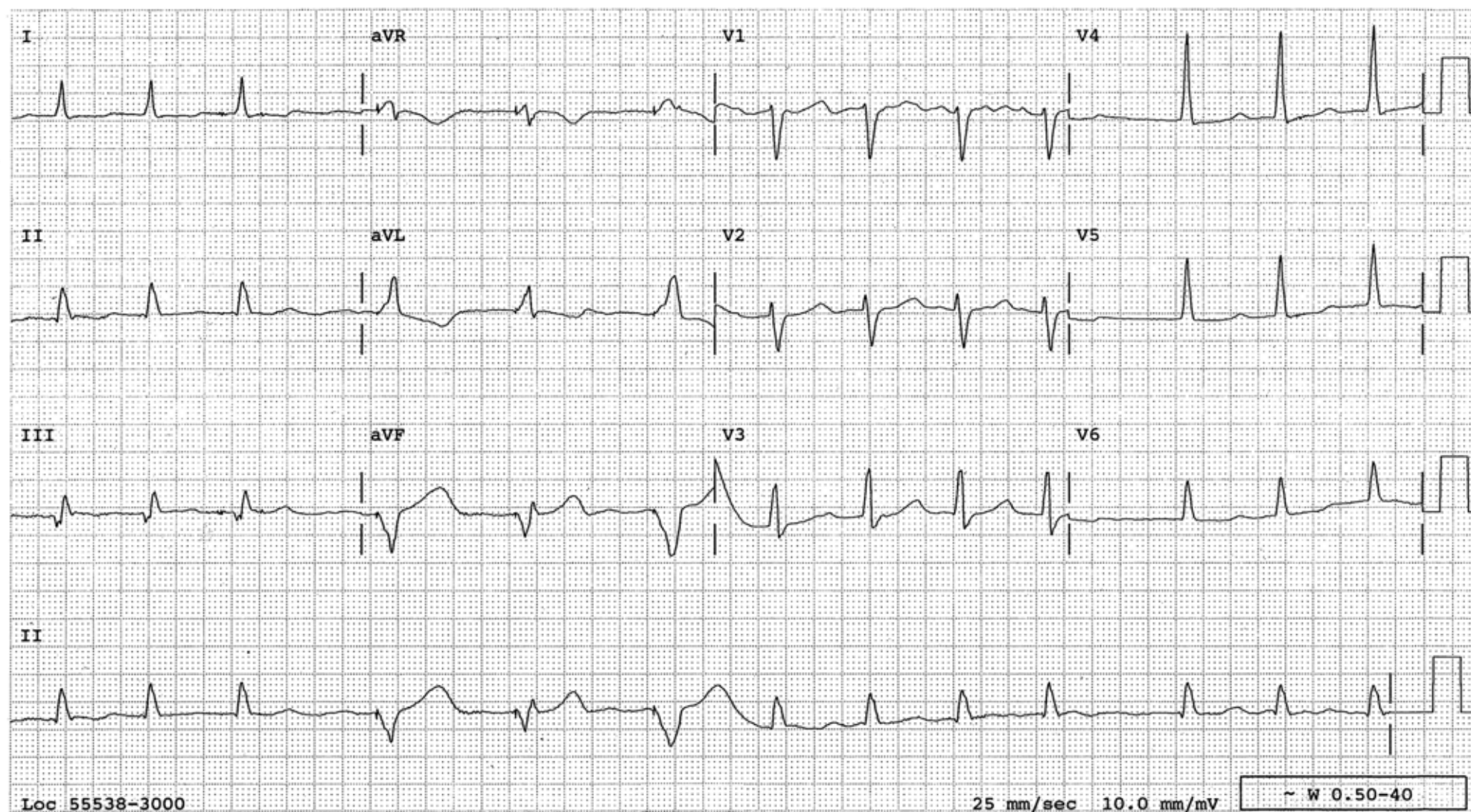
- Dx:*
1. Sinus rhythm
 2. Multiple PACs, some are conducted and some are not conducted, resulting in pauses
 3. Nonspecific ST-T changes



7-9

- 7-9 A regular rhythm with occasional pauses. These pauses are exactly two times the basic cycle length. There are no P waves that are blocked indicating that there is no AV block. This is an example of SA block. SA block without any change in the R-R interval indicates that there is no progressive lengthening of the SA conduction time prior to the block, hence Type II SA block. In Type I SA block, the progressive lengthening of the SA conduction time prior to the block will cause the pause to be shorter than two R-R intervals.

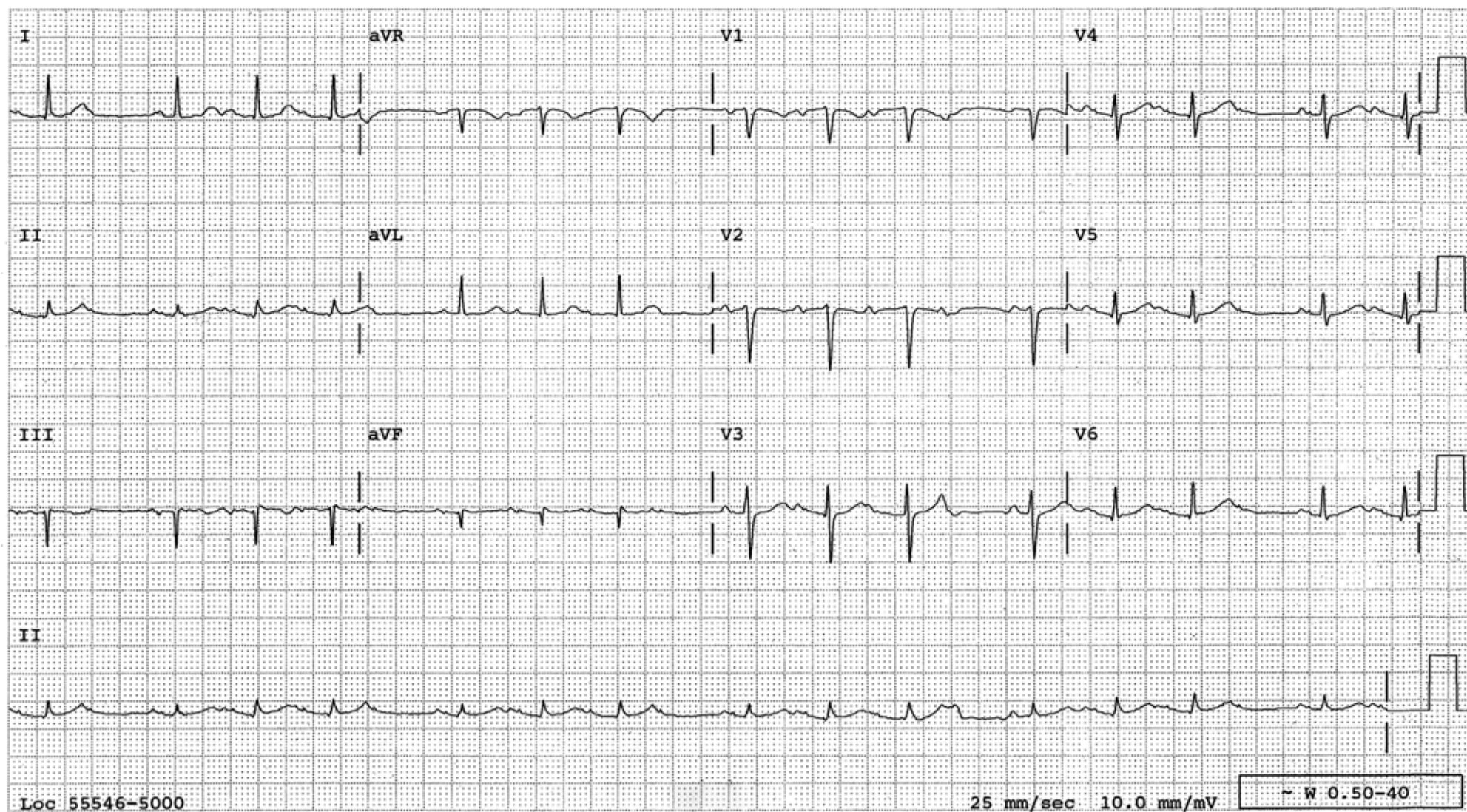
Dx: Sinus rhythm with occasional Type II SA block



7-10

7-10 Atrial fibrillation with a ventricular response of about 80/minute. Occasionally, the ventricular rate slows down allowing the artificial demand pacemaker to take over at a rate of 60/minute. Of the three paced beats, the QRS morphology of the middle one is different from the other two. The middle one is a fusion of paced and natural beats. Q waves are wide and deep in lead III reflecting old inferior infarct.

- Dx:*
1. Atrial fibrillation
 2. Normally functioning artificial demand pacemaker with one fusion beat
 3. Old inferior infarct



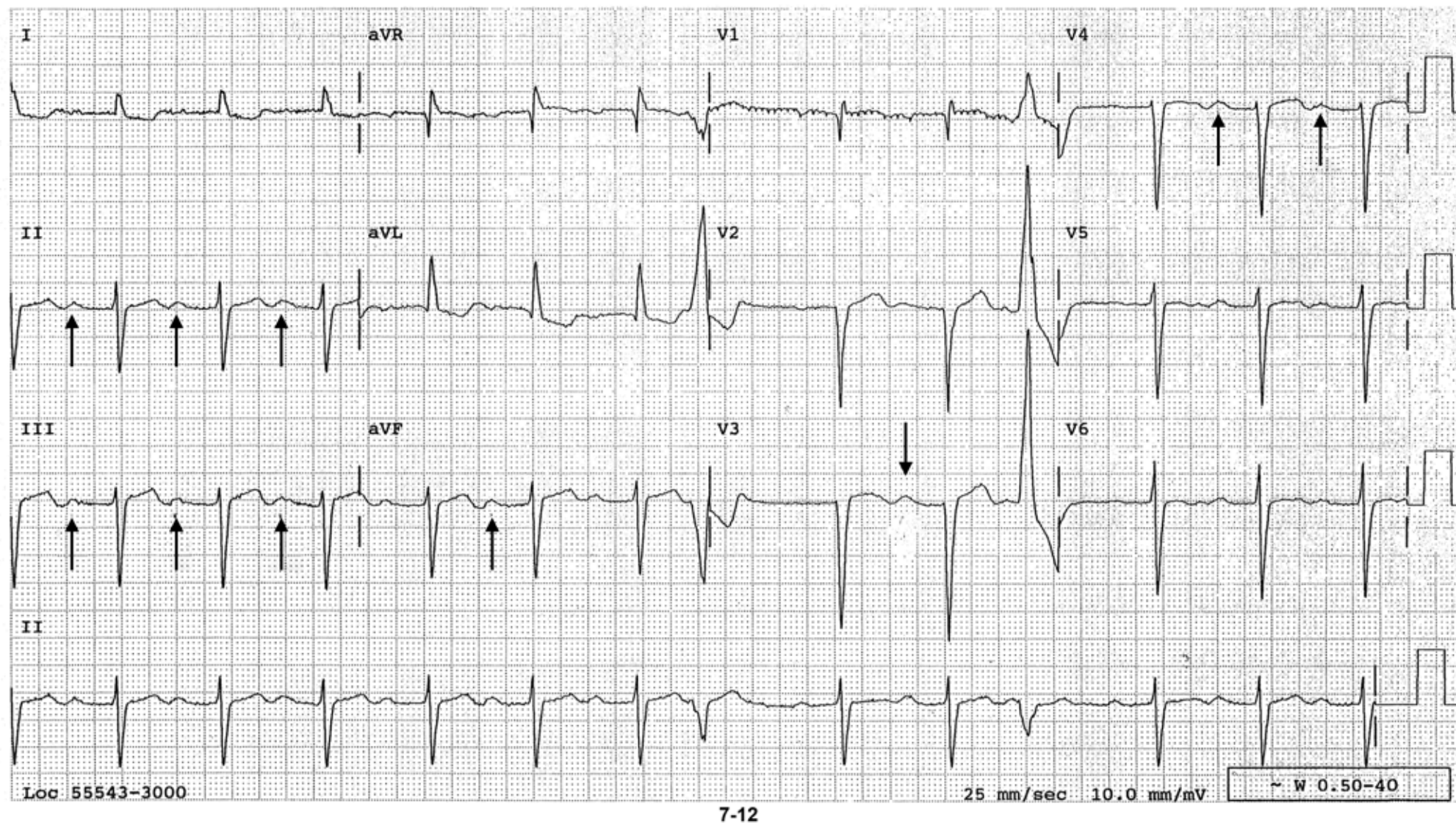
7-11

7-11 There is a group beating of the QRSs (three QRSs are grouped together). Careful examination, especially of V_2 , reveals progressively lengthening P-R interval until the fourth P wave is blocked revealing 4:3 AV Wenckebach phenomenon. QS pattern in leads III and aVF with a small Q wave in lead II indicate an old inferior infarct.

- Dx:
1. Sinus tachycardia with 4:3 AV Wenckebach phenomenon
 2. Old inferior infarct

7.12 Question: The deflections pointed by arrows (↑) are (choose one from below):

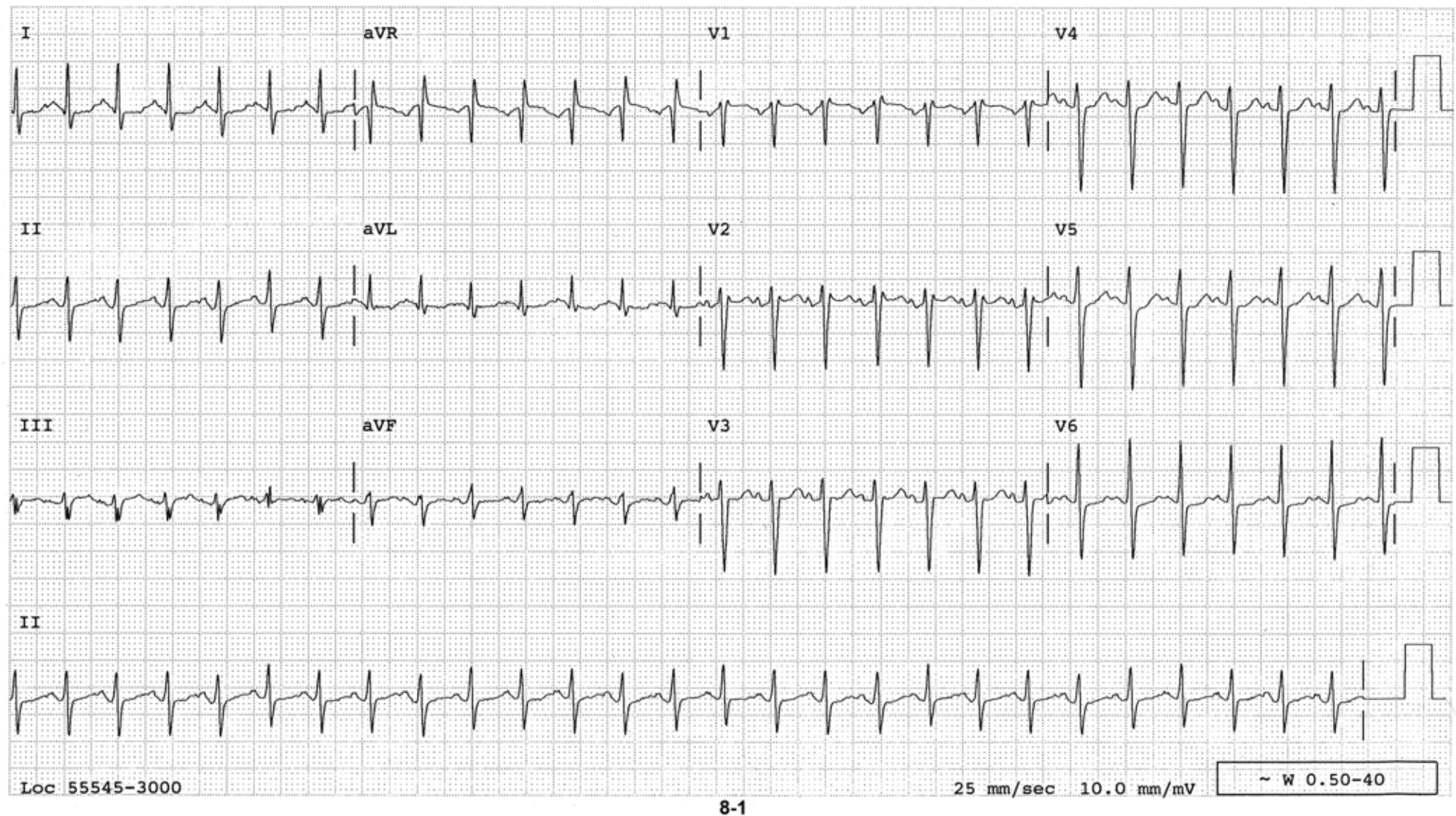
1. P waves only
2. U waves only
3. Summation of P and U waves



Answer: 3. Summation of P and U waves

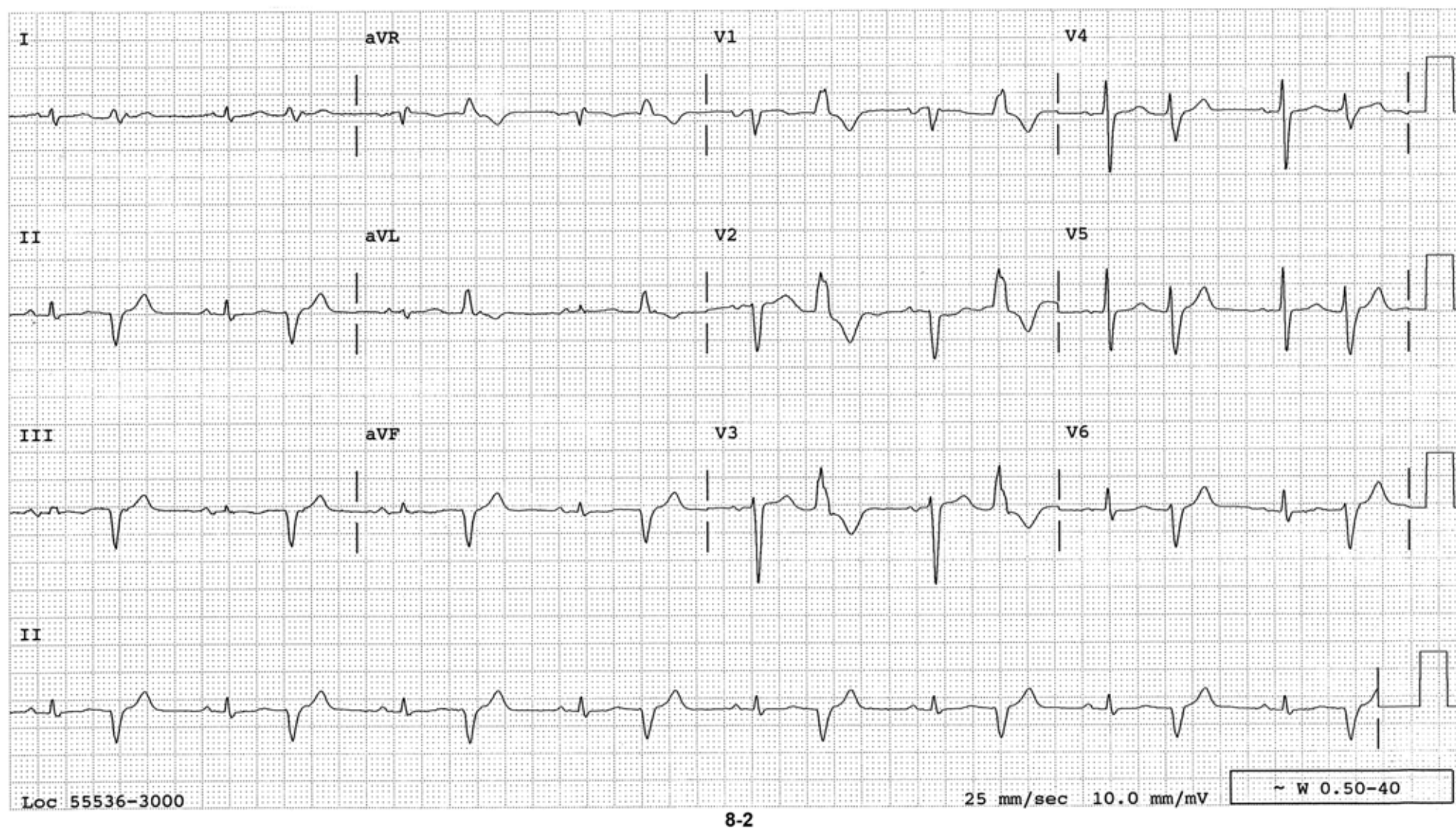
Discussion: The deflections pointed by arrows (↑) can pass as P waves with 1°AV block or U waves or both. Luckily there are two PCVs. The compensatory pause after the PVC in the rhythm strip of lead II clearly reveals a P wave with 1°AV block. Could a U wave be superimposed on the P wave at other times? This question is solved by the fact that, as the luck would have it, the P wave in V₃ in this patient is isoelectric judging from the fact that the compensatory pause after the PVC is flat. That means the deflection pointed by the arrow (↓) has to be a U wave, proving that the deflections pointed by the arrows (↑) are P and U waves together. Why is it important to sort it out? If this is a patient with an infective endocarditis of the aortic valve, the 1°AV block can be the first or the only sign of paravalvular abscess. Prominent U wave may be the reflection of hypokalemia, which is important to correct if you are seeing this patient for preoperative consultation. If it is U wave only without a P wave, the rhythm is accelerated AV junctional rhythm and one has to consider digitalis intoxication if the patient is taking digitals or myocardial ischemia or infarction or excess amount of catecholamines circulating, which occurs in any stressful condition. At times PVCs can be very useful in helping us interpret an ECG as happens in this case.

SECTION 8



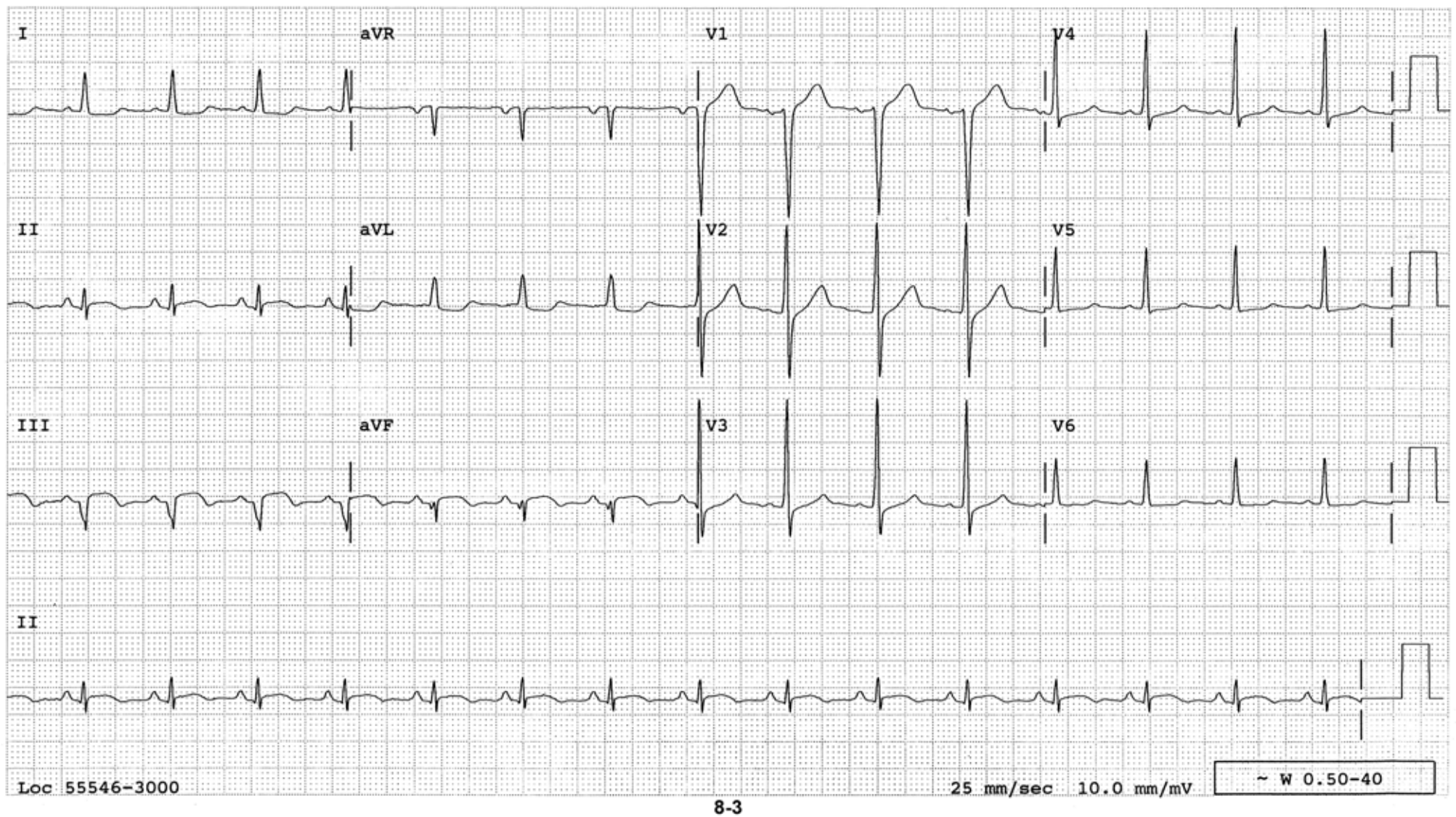
8-1 A regular narrow QRS tachycardia at a rate of 161/minute. There is a P wave in front of each QRS and the rhythm can be sinus tachycardia or ectopic atrial tachycardia. The P wave morphology in leads VI and II will settle the issue. The P wave during sinus rhythm is biphasic (initially positive, then negative) in V_1 with or without LAE and is entirely upright in lead II. Since that is the case in this tracing, this is sinus tachycardia. Mean QRS axis is shifted to the left reflecting LAFB. The transition occurs in V_6 . Late transition is common in LAFB.

- Dx:
1. Sinus tachycardia
 2. LAFB
 3. Late transition



8-2 Sinus rhythm is present. Every other complex is ventricular premature complex, hence ventricular bigeminy. The P wave is mostly negative in V_1 indicating LAE. QRS voltage is abnormally low in the limb leads. R waves do not progress normally in the right precordial leads raising a possibility of old anteroseptal infarction.

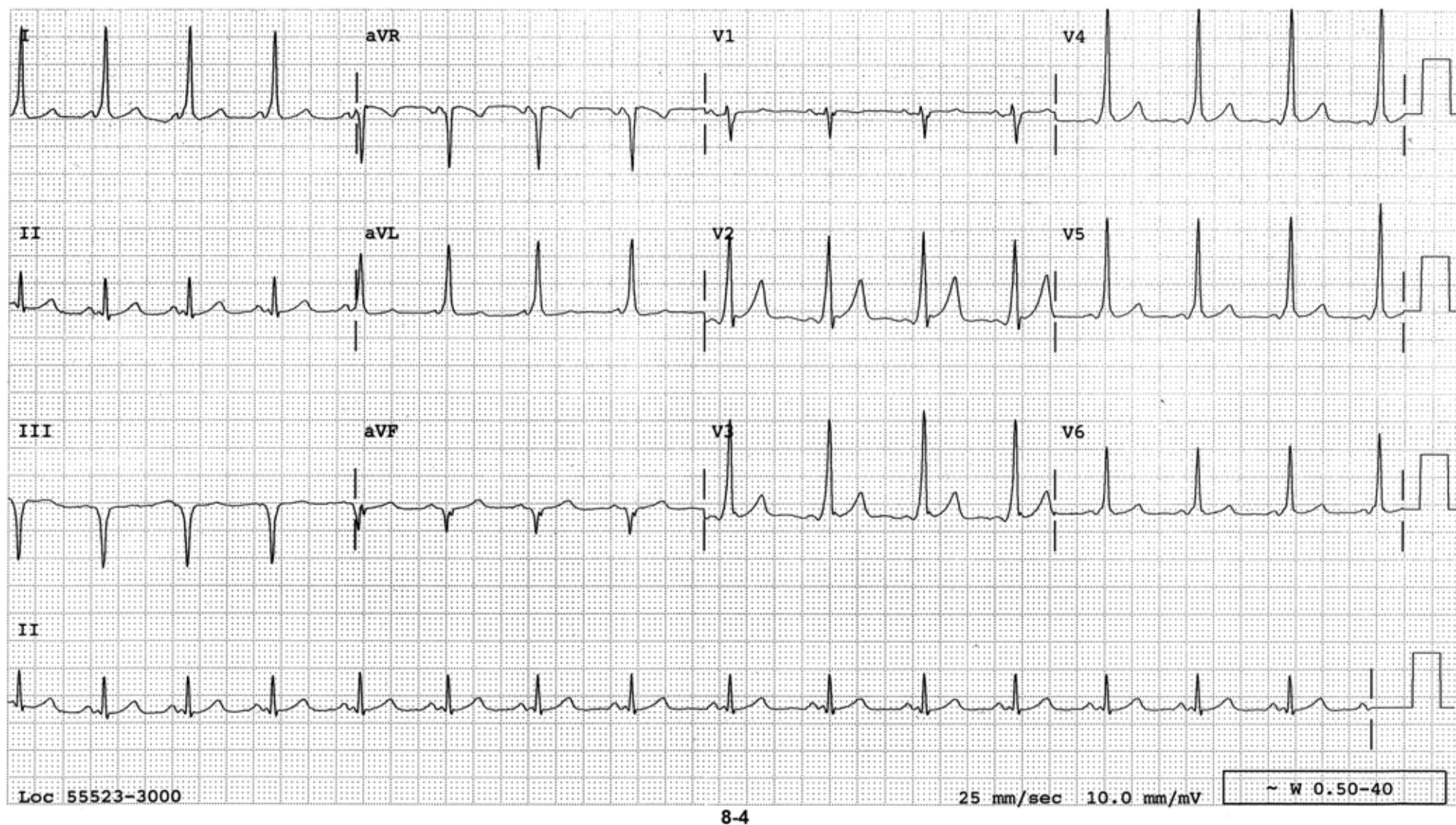
- Dx:
1. Sinus rhythm with ventricular bigeminy
 2. LAE
 3. Low voltage in the limb leads
 4. Poor R wave progression



8-3 Sinus rhythm at a rate of 92/minute. QS pattern in leads III and aVF with ST elevation and tall R waves in leads V_2 and V_3 reflect inferoposterior infarct, probably recent.

Dx: 1. NSR

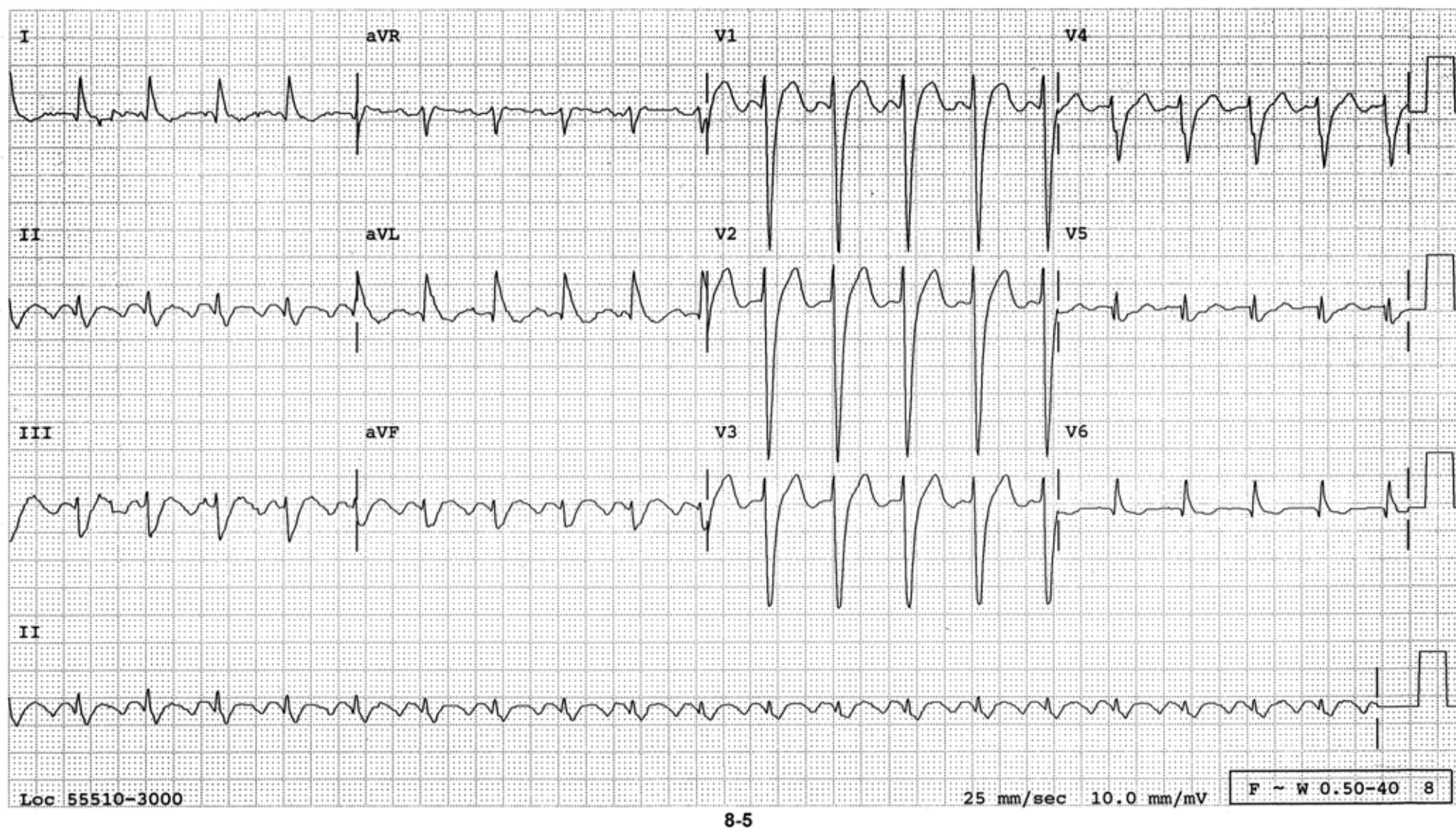
2. Inferoposterior infarct, probably recent



8-4 Normal sinus rhythm at a rate of 91/minute. This tracing also suggests inferoposterior MI, but the P-R interval is short and the upstroke of the QRS is slurred in many leads, and are diagnostic of WPW syndrome. The delta wave is oriented superiorly and anteriorly registering as a negative wave in the inferior leads and tall R waves in V_2 - V_3 , simulating inferoposterior MI.

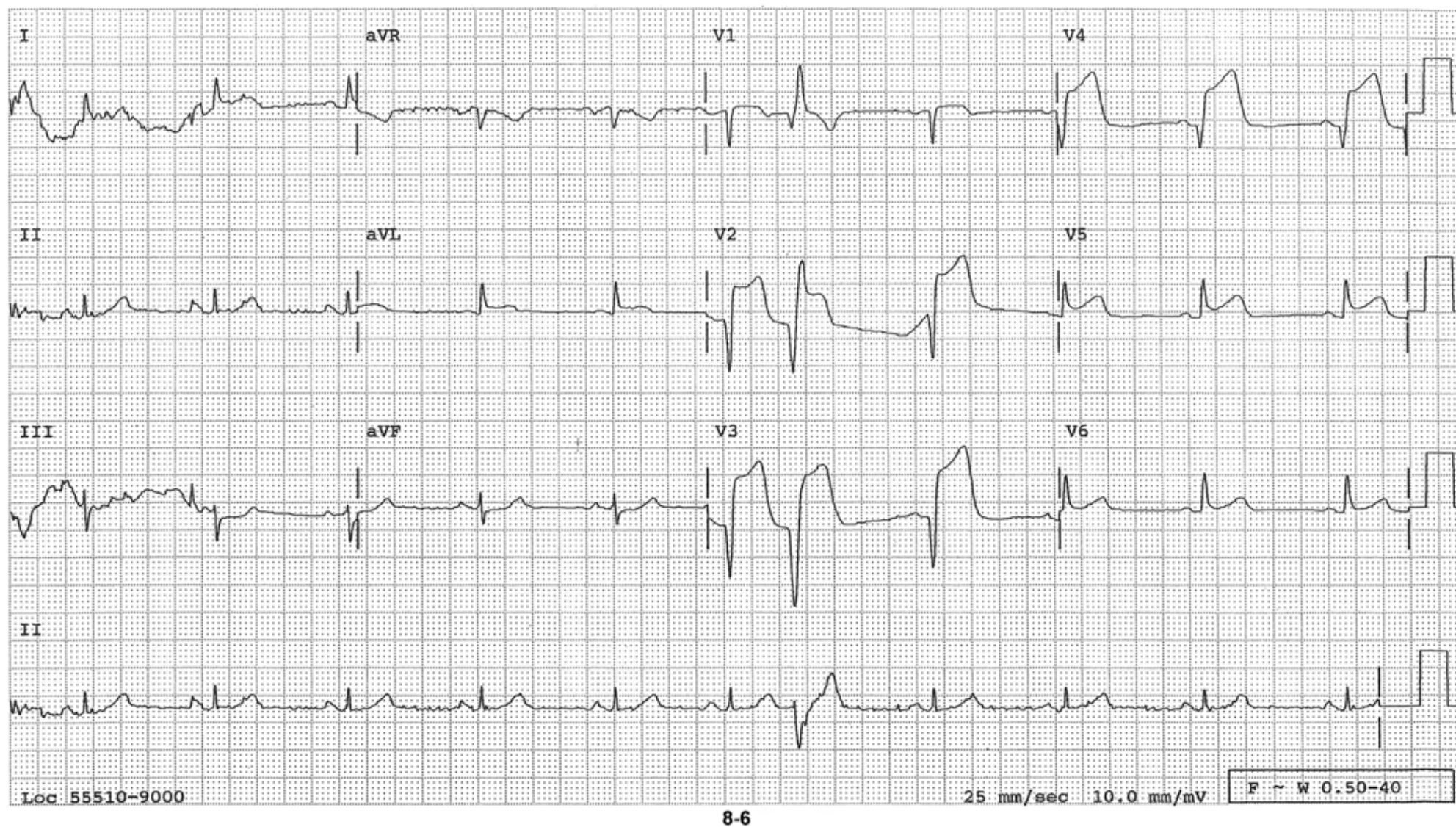
The P-R interval is normal in V_1 and no definite delta wave can be appreciated. This is because the delta wave is isoelectric in this lead.

- Dx:
1. NSR
 2. WPW syndrome simulating inferoposterior infarct



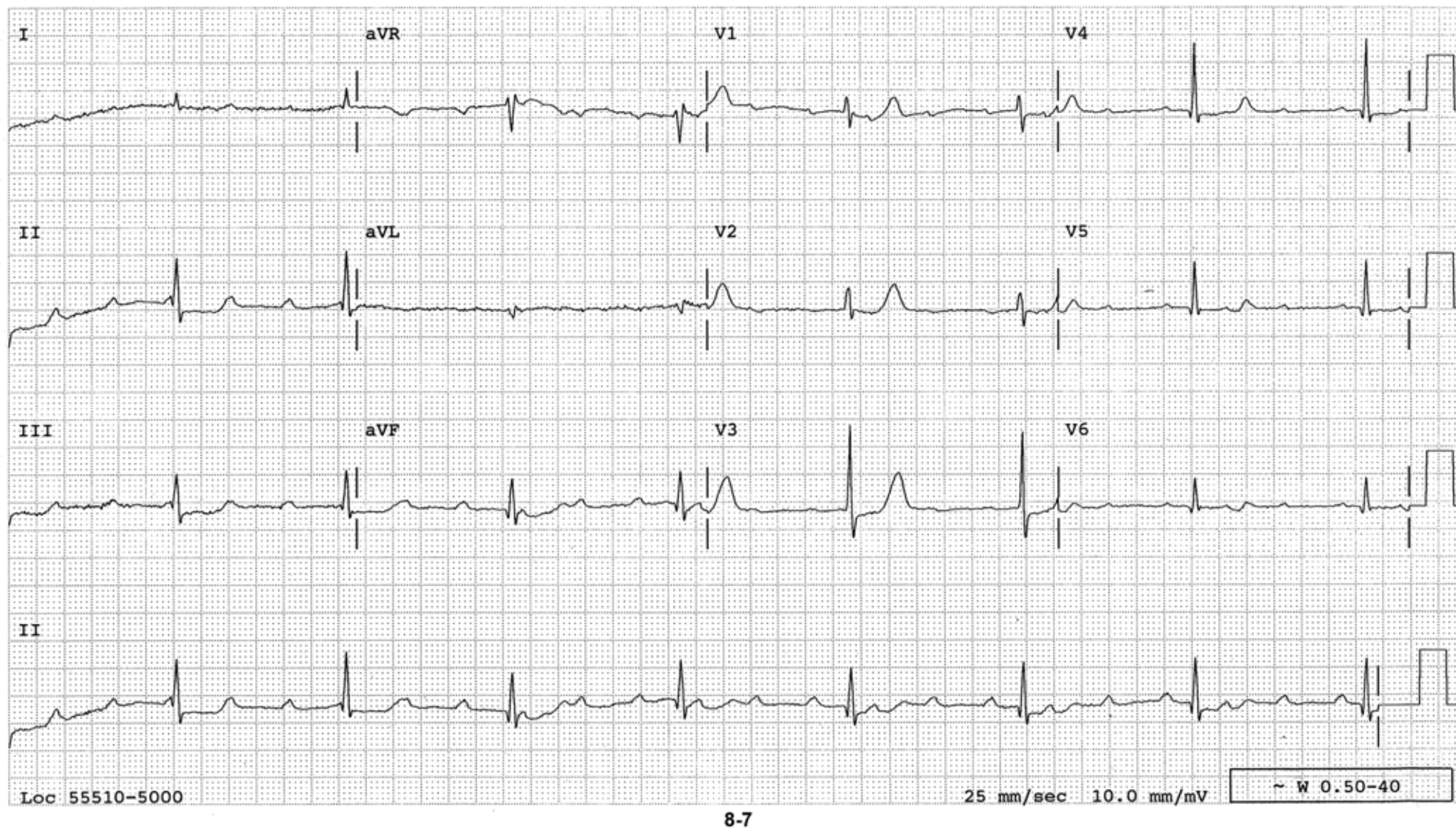
8-5 A regular rhythm at a rate of 120/minute. The QRS is wide and measures 140 milliseconds. The findings in V_1 suggest sinus tachycardia. However, careful examination of the rhythm strip of lead II reveals the "domes" of flutter waves. Every other dome is deformed by the QRS making the recognition of the domes difficult. The flutter rate is 240/minute which is somewhat slow. Type Ia or Ic antiarrhythmic agents can easily slow down the flutter rate to this level. Voltage criteria and ST-T changes for LVH are present.

- Dx:
1. Atrial flutter with 2:1 AV conduction
 2. IVCD
 3. LVH



8-6 Normal sinus rhythm with one PVC. Acute antero-septal STEMI is present. The PVC in V_1 - V_2 has a QR pattern with ST elevation, effectively revealing the STEMI as well. Thus, QRSs originating from the ventricle such as PVCs, QRSs of VT, accelerated idioventricular rhythm or ventricularly paced rhythm can at times effectively reveal STEMI (it has to be a QR or qR pattern). If the patient has LBBB masking STEMI, PVCs revealing STEMI can be very useful.

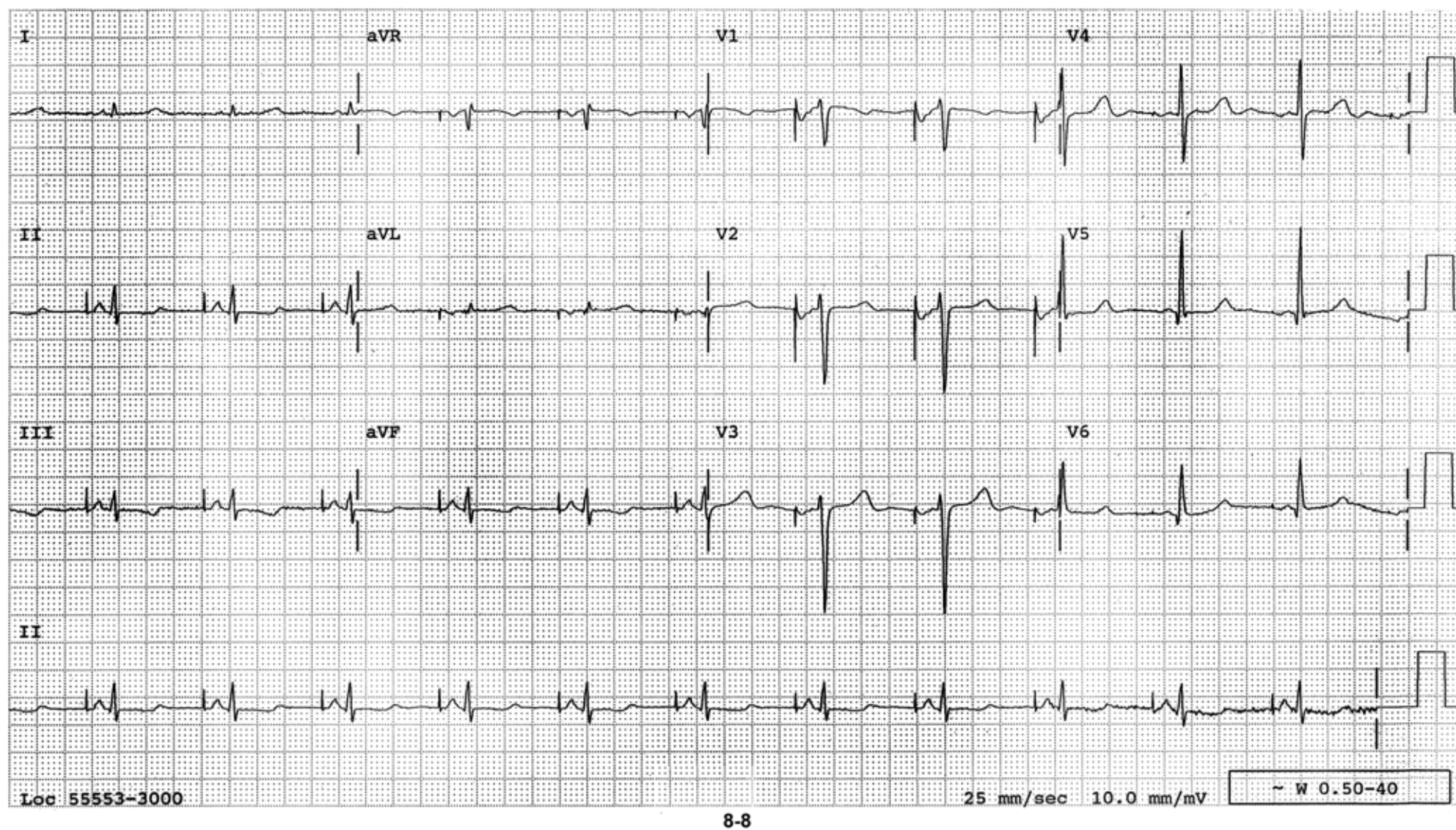
- Dx:*
1. NSR
 2. Antero-septal STEMI
 3. One PCV, which also reveals STEMI



8-7 A regular rhythm at a rate of 49/minute. QRSs are narrow. P waves occur regularly but they have no fixed relationship to the QRSs and there are more P waves than QRSs indicating complete (3°) AV block. R waves are tall in the right precordial leads with a slight horizontal ST depression, highly suggestive of acute posterior myocardial infarct which may be the cause of this 3° AV block.

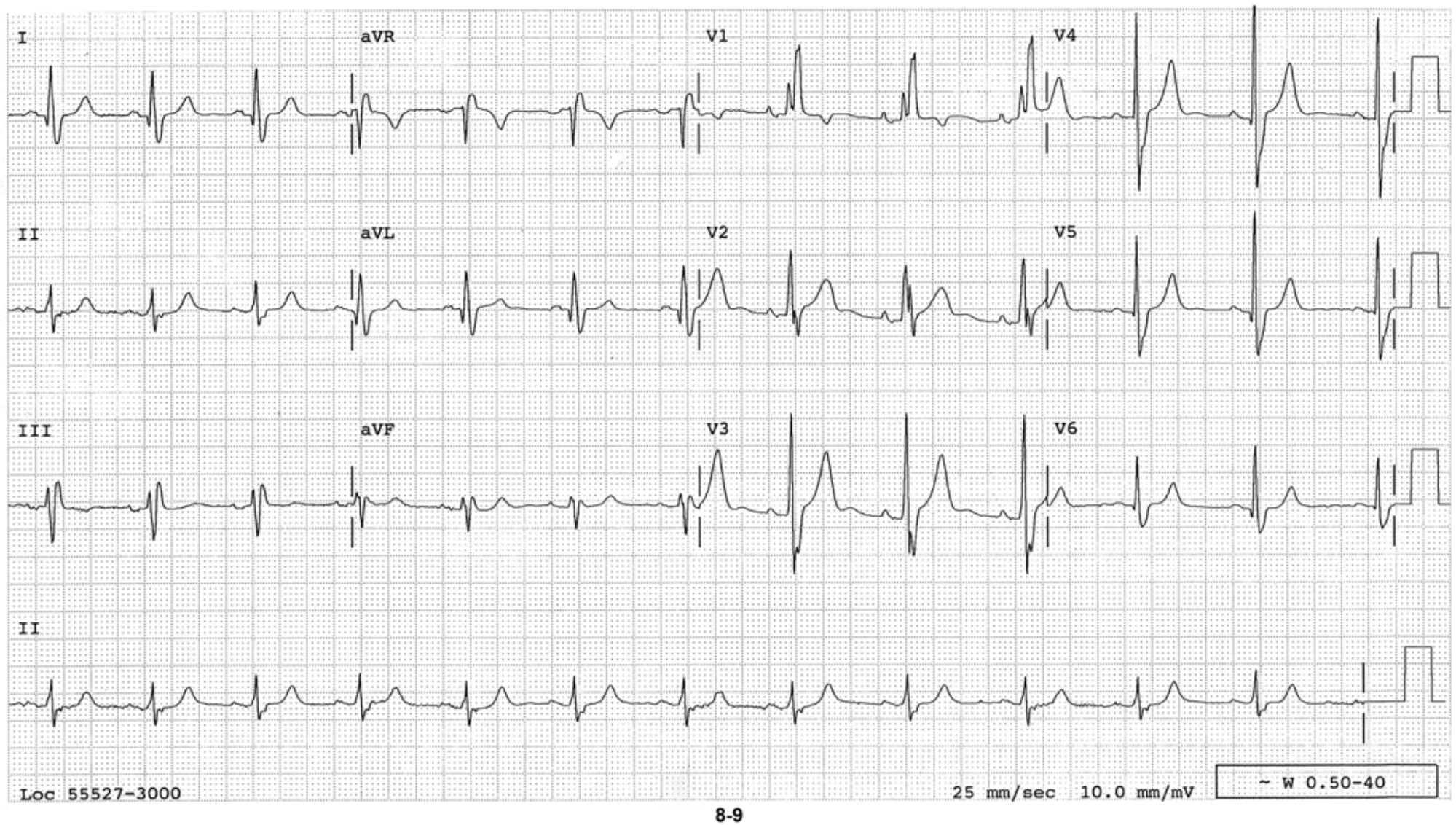
Dx: 1. Sinus rhythm with 3° AV block

2. Acute posterior myocardial infarct is quite likely, which may be the cause of 3° AV block



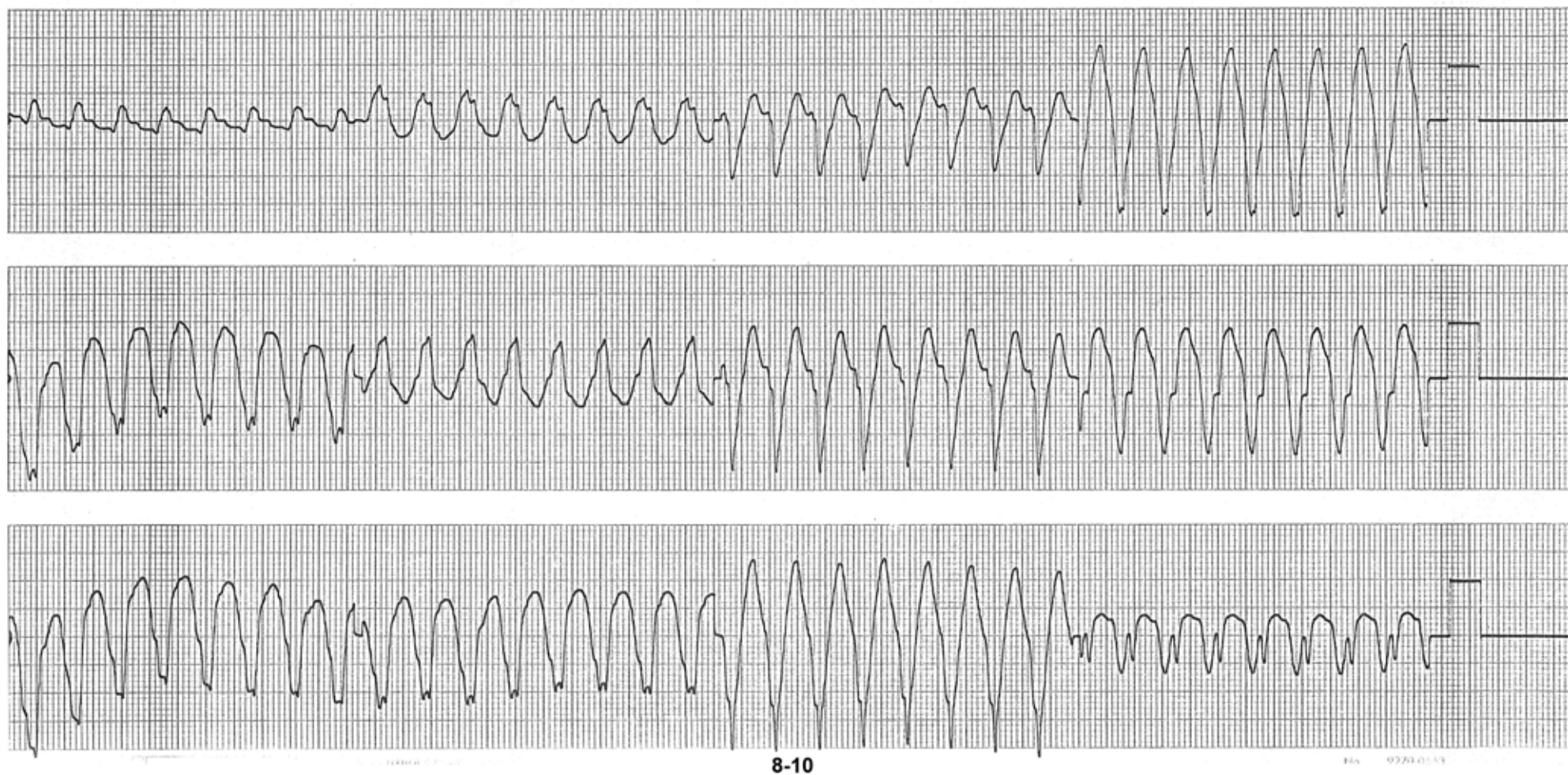
8-8 Artificial pacemaker spikes are readily recognizable which is pacing the atrium. After the P wave, a narrow QRS occurs consistently without a pacemaker spike in front of it indicating that this is an atrial paced rhythm. T waves are inverted in the inferior leads.

- Dx:*
1. Atrial paced rhythm
 2. Nonspecific inferior T waves changes



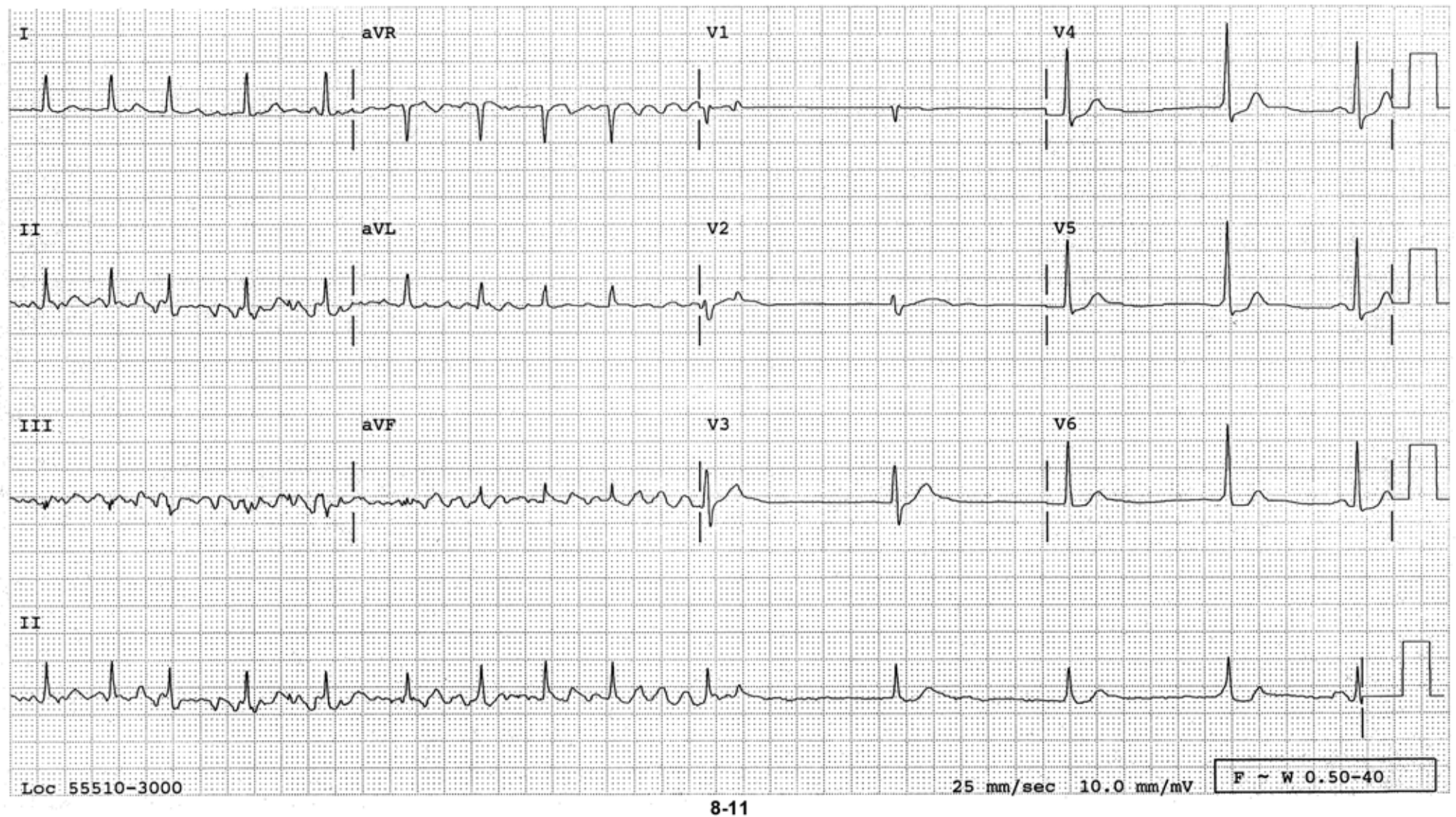
8-9 Normal sinus rhythm at a rate of 75/minute. The QRSs are wide and measures 124 milliseconds. rsR' in V₁ and broad S waves in leads I, aVL and V₆ are typical of RBBB.

- Dx: 1. NSR
2. RBBB



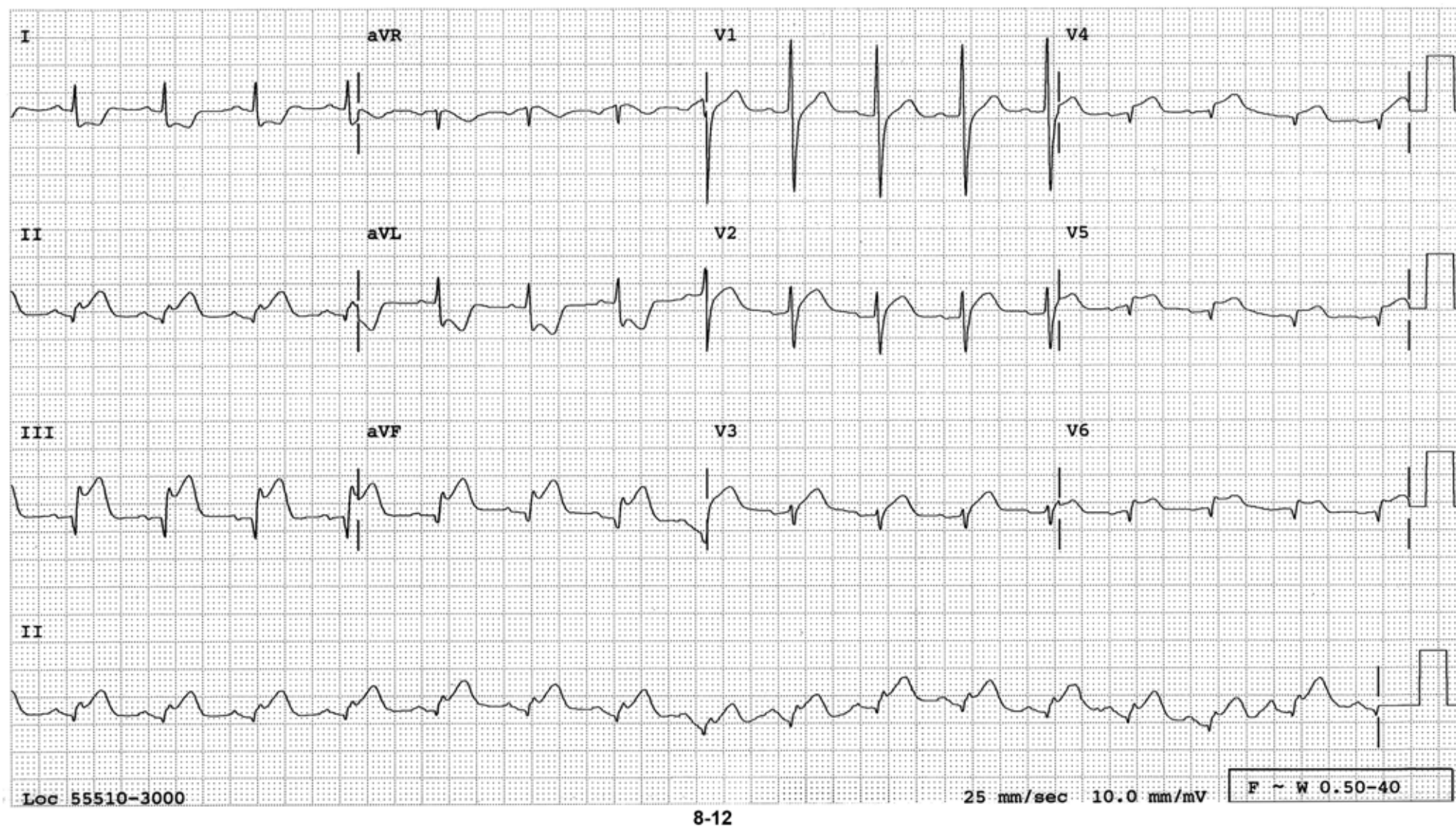
8-10 A regular wide QRS tachycardia at a rate of 192/minute. There is a concordance of the QRSs in the precordial leads (QRSs pointing down in every precordial leads, called negatively concordant) which is very good for VT. Also, QS pattern in V_6 is diagnostic of VT, i.e. QS pattern in V_6 means that the ventricular depolarization originated there and is propagating away from there. What is under V_6 ? Ventricular apex. Therefore the rhythm is a VT.

Dx: VT



8-11 Atrial fibrillation is present in the first half of the tracing. This rhythm is spontaneously terminated allowing the AV junctional pacemaker to escape for three beats until eventually a sinus P wave occurred in time to capture the ventricle.

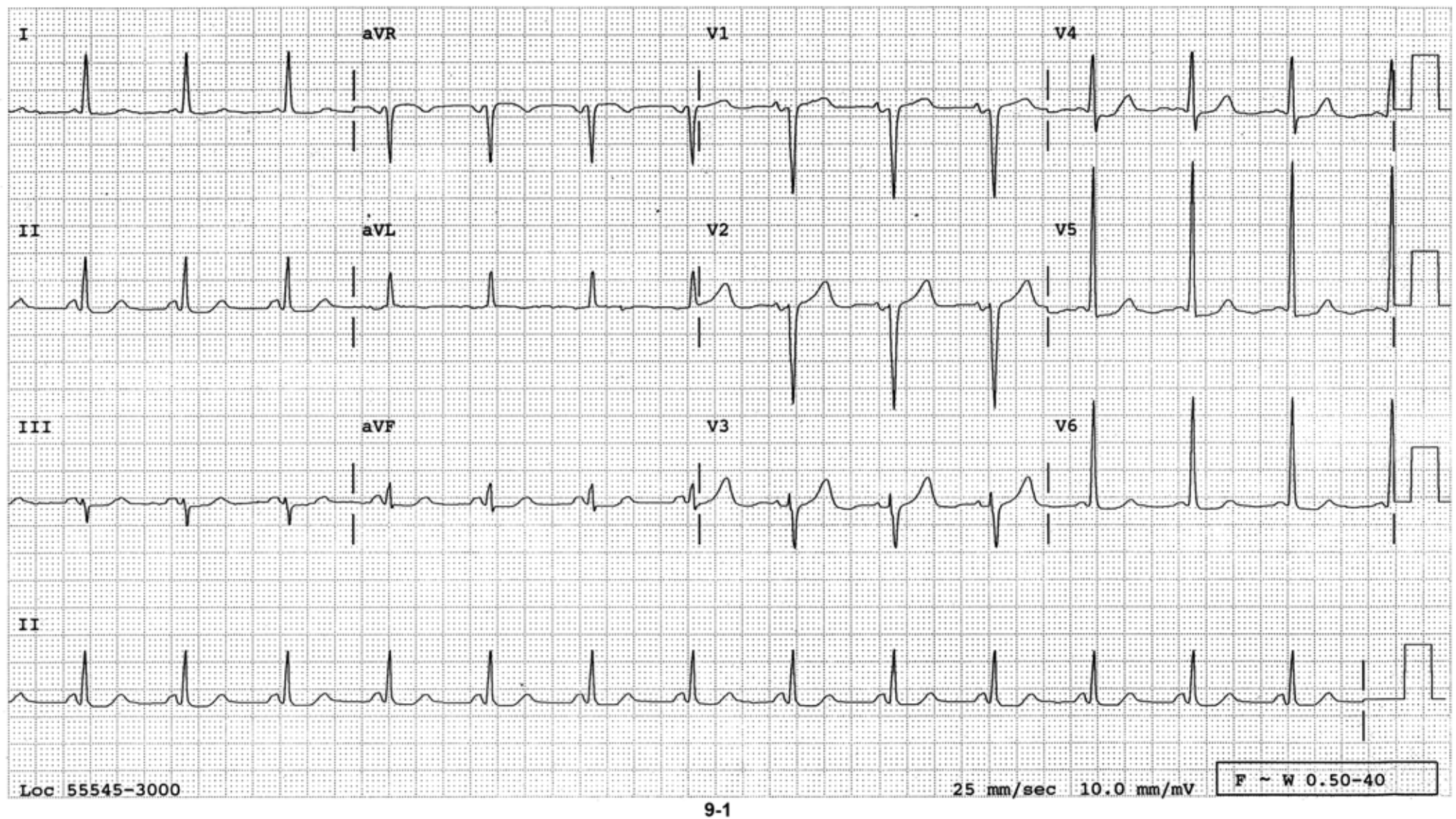
Dx: Atrial fibrillation with spontaneous termination, followed by AV junctional escape beats and then eventually a sinus beat



8-12 An NSR at a rate of 90/minute. ST elevation with Q waves in the inferior leads indicates acute inferior MI. Leads V_4 - V_6 also reveal Q waves with ST elevation. The R waves are tall in V_1 . These findings raise the possibility of inferoposterolateral infarction. However, the P waves in V_4 - V_6 are either biphasic or entirely negative indicating that these are not regular left-sided precordial leads. (In regular left-sided precordial leads, the P waves in these leads should be entirely positive). Besides, leads I, aVL and V_6 look at the heart from similar angles and should have similar looking QRSs. In this tracing, the QRSs in I and aVL are similar, but the one in V_6 is entirely different. These two clues indicate that these are right-sided precordial leads. ST elevation in these right-sided precordial leads indicates RV infarction. This can be already predicted by ST depression in lead I in addition to lead aVL, which means the ST vector is pointed down and to the right. Why to the right? Because the RV is involved. In inferior MI, if RV is not involved, the ST vector is pointed more or less straight down, which is perpendicular to lead I axis and the ST-segment will not be deviated in lead I.

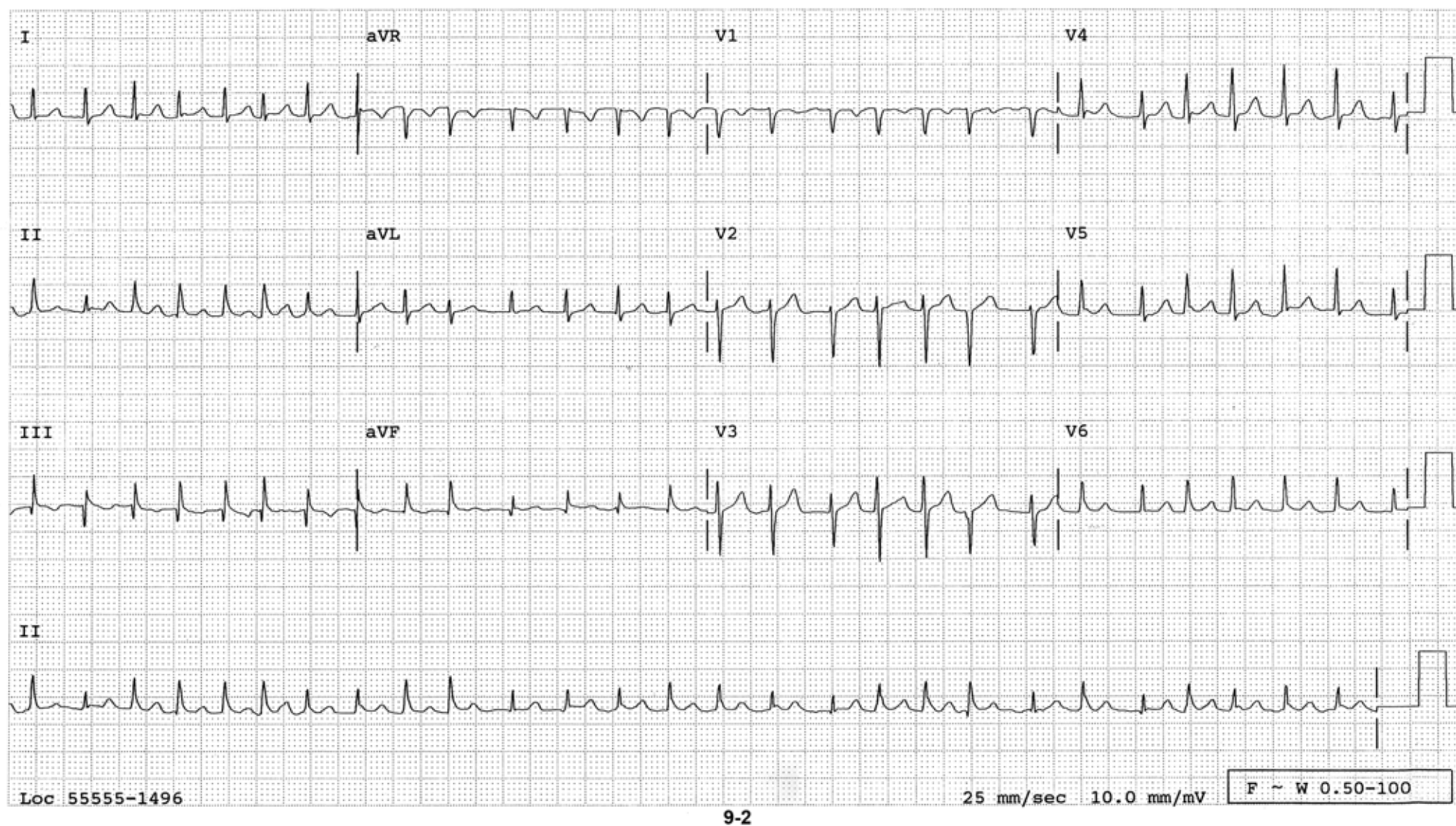
- Dx: 1. NSR
2. Acute inferior infarct involving the RV

SECTION 9



9-1 Normal sinus rhythm at a rate of 81/minute. The P-R interval is short and measures 110 milliseconds. However, there are no distinct delta waves in any of the 12 leads and this is not an example of WPW syndrome. This will be called accelerated AV conduction ("greasy" AV node). This short P-R interval is now considered to be one end of normal spectrum of P-R interval. Voltage criteria and mild ST changes for LVH are present.

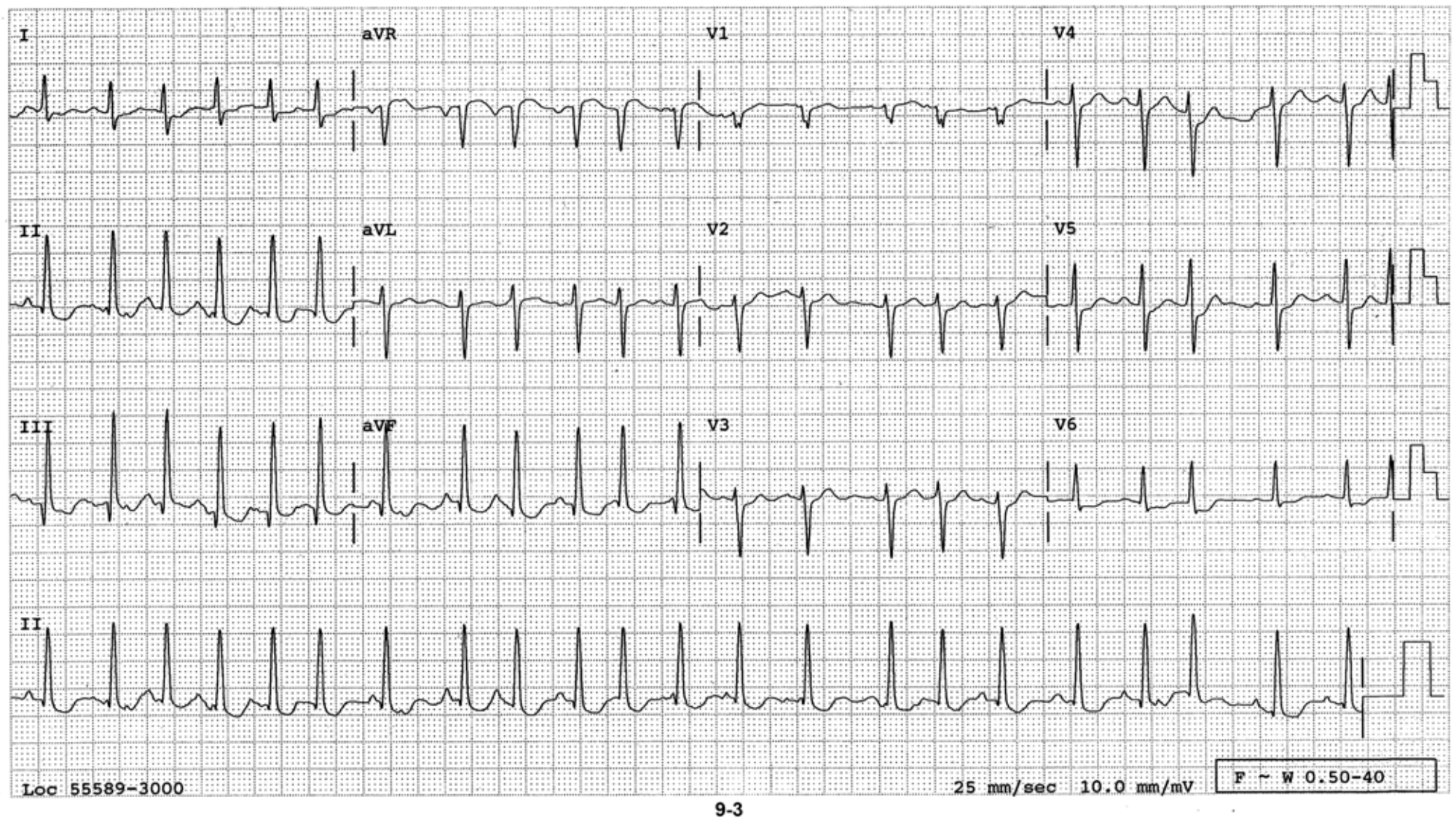
- Dx:
1. NSR
 2. Accelerated AV conduction
 3. LVH



9-2

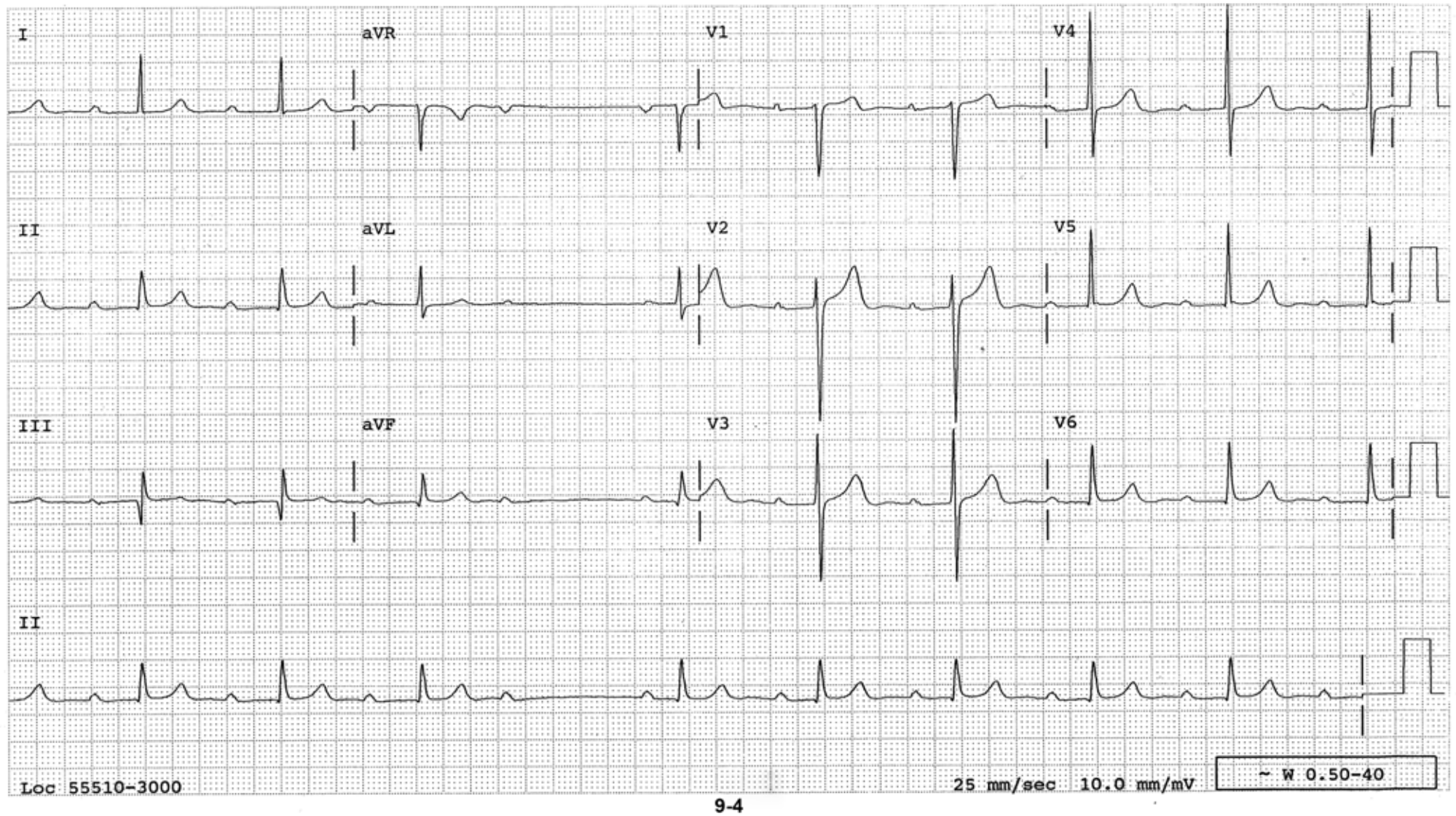
9-2 An irregularly irregular rhythm with narrow QRS complexes, at a rate of 166/minute. No distinct P waves are recognized and the rhythm is atrial fibrillation. The Q wave in lead III is deep, but not wide enough to make one think of inferior infarction.

Dx: Atrial fibrillation with ventricular rate of 166/minute



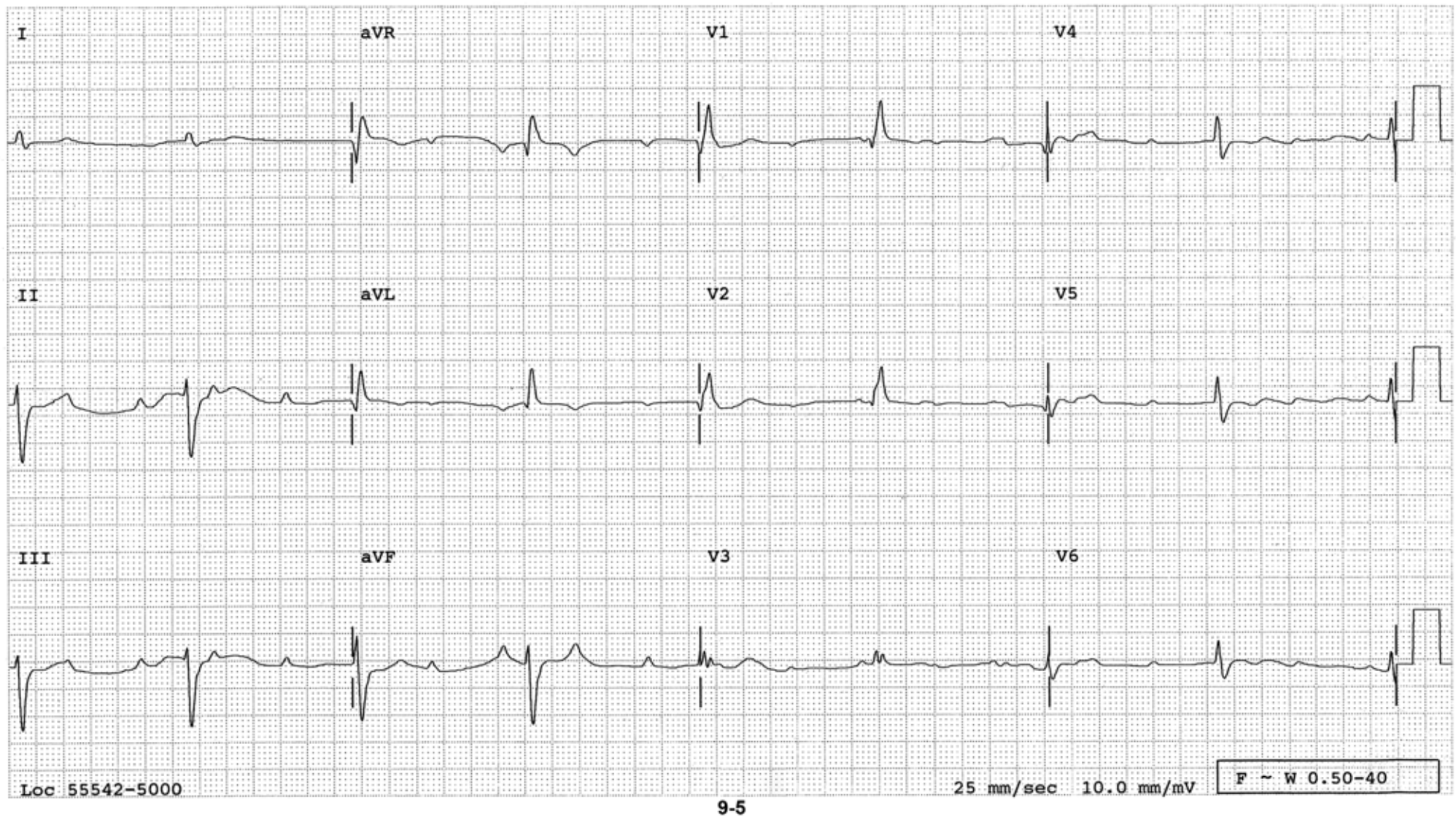
9-3 Irregularly irregular rhythm at a rate of 140/minute is present. The QRSs are narrow. There is a P wave in front of each QRS. These P waves occur irregularly with changing morphology. These are all diagnostic features of MAT. There is a late transition. Voltage criteria and ST-T changes for LVH are present (note the precordial leads are half-standard).

- Dx:*
1. MAT, 140/minute
 2. Late transition
 3. LVH



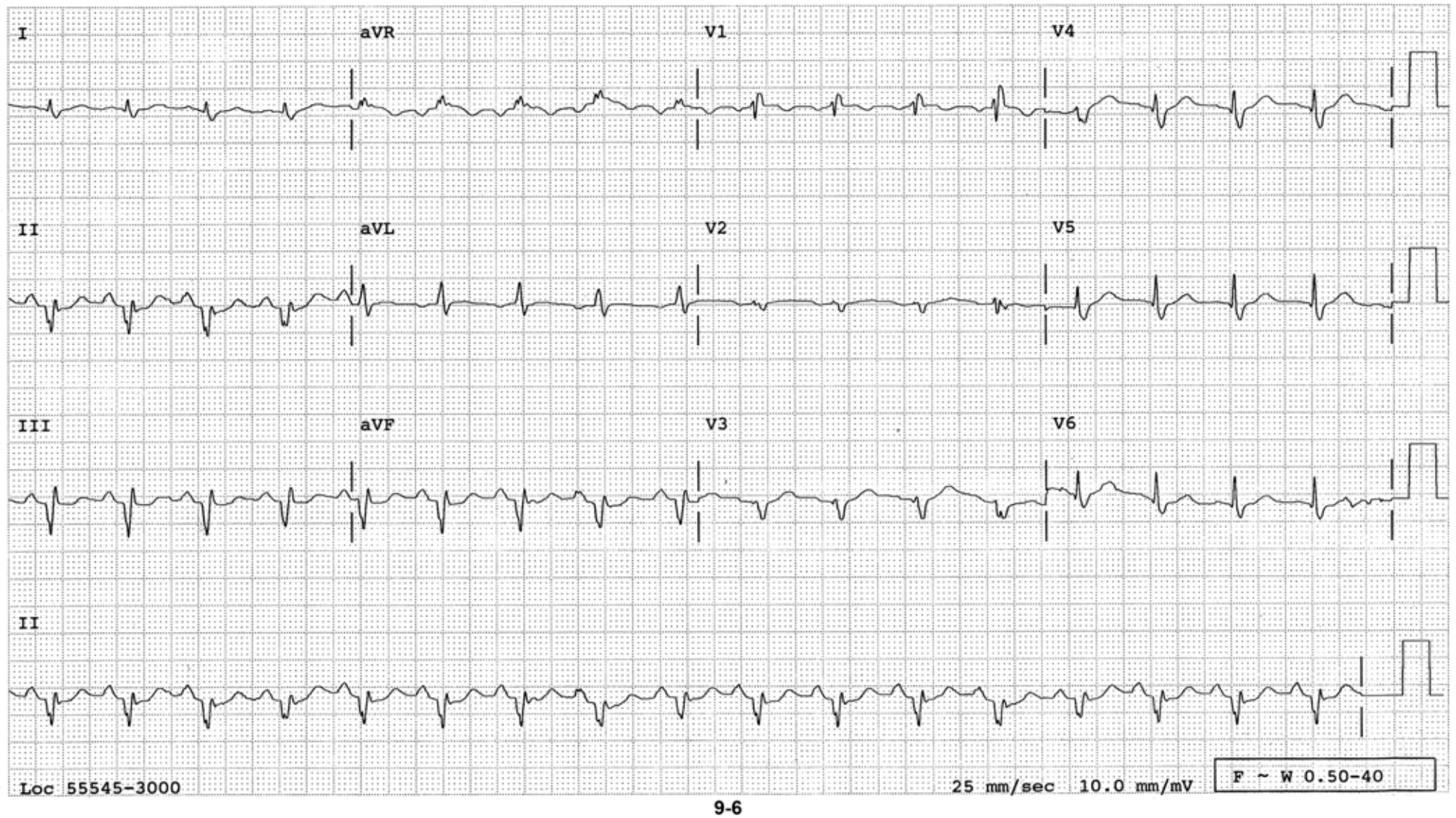
9-4 P waves occur regularly at a rate of 60/minute. The P waves are occasionally blocked. The P-R interval progressively lengthens before this happens, and is diagnostic of AV Wenckebach phenomenon. Q waves are prominent in lead III suggesting old inferior MI.

- Dx:
1. Sinus rhythm with Type I 2° AV block
 2. Probable old inferior infarct



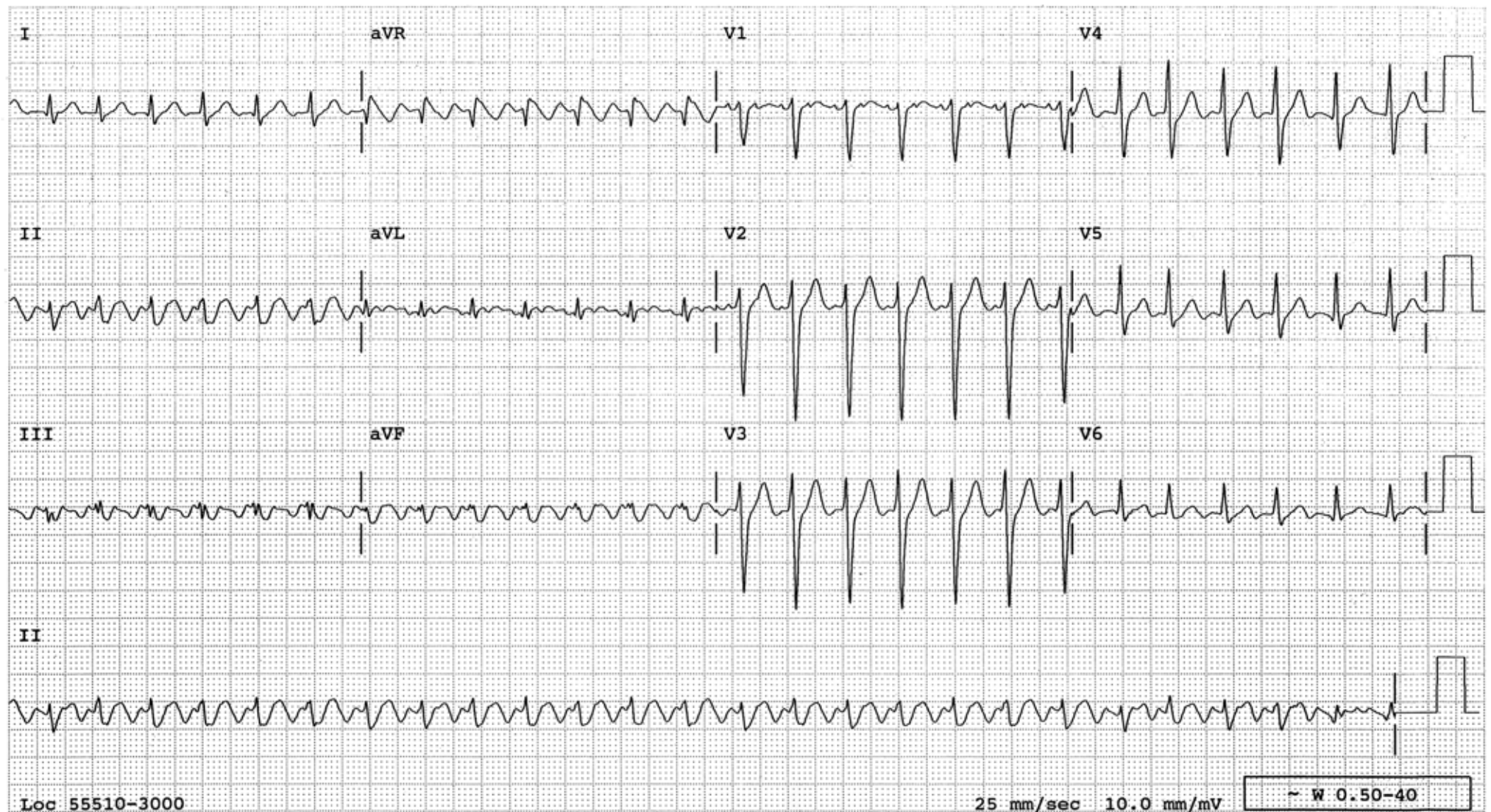
9-5 QRSs occur regularly at a rate of 48/minute. The P waves and the QRSs do not have a fixed relationship indicating complete AV block. The QRSs are wide raising the possibility of either ventricular escape rhythm or AV junctional escape rhythm in a patient who has preexisting conduction defect (BIFB). An old tracing will be useful in sorting out these two possibilities.

Dx: 3° AV block with either ventricular escape rhythm or junctional escape rhythm with preexisting BIFB (RBBB and LAFB)



9-6 Sinus tachycardia at a rate of 103/minute. P waves are mostly negative in V_1 indicating LAE. rsR' pattern in V_1 and S waves in leads I, aVL, V_6 with QRS duration measuring only 114 milliseconds make incomplete RBBB. Q waves are deep and wide in the inferior leads indicating old inferior infarct. R waves do not progress normally in the right precordial leads suggesting old antero-septal infarct as well.

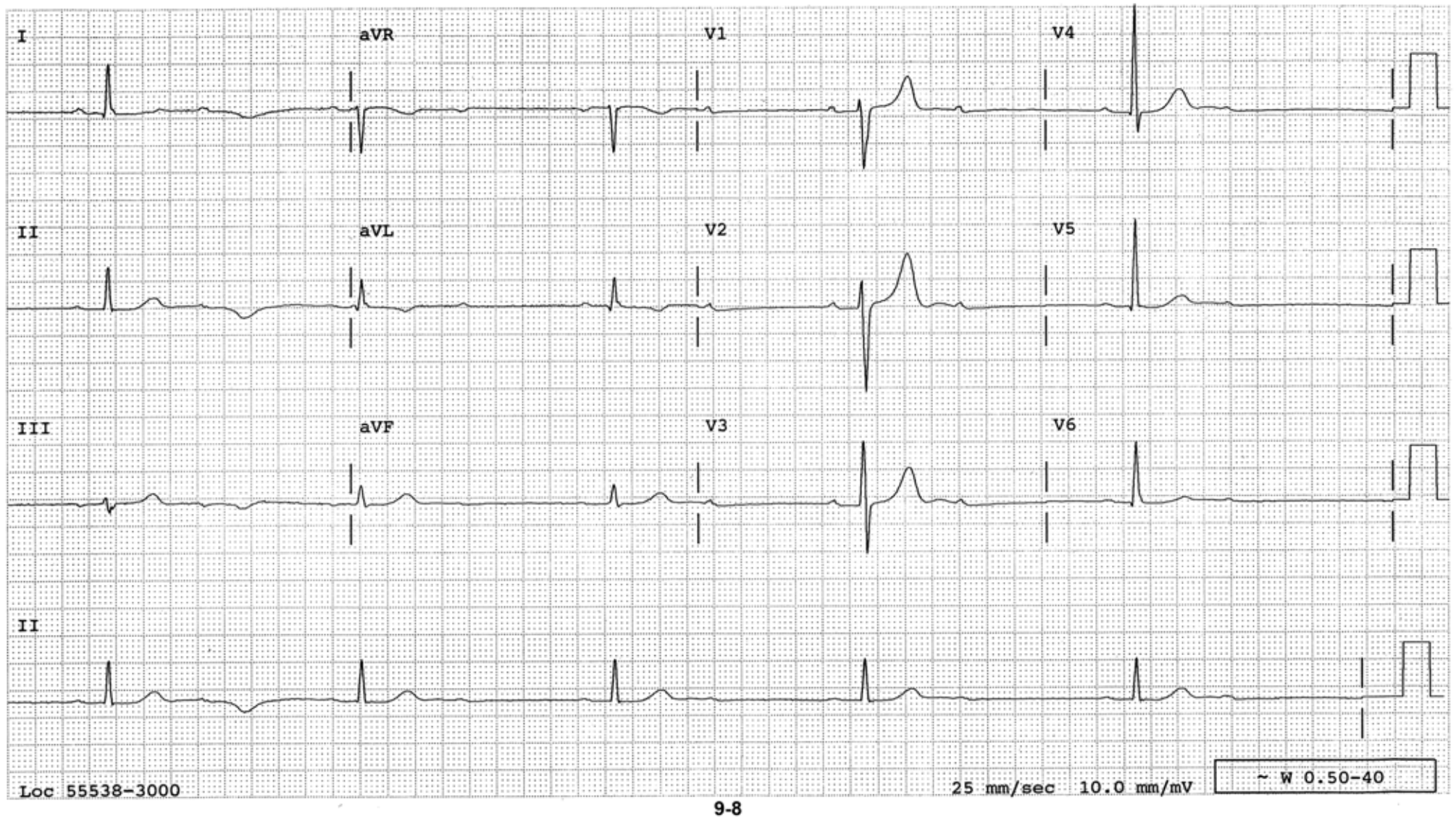
- Dx:*
1. Sinus tachycardia
 2. Incomplete RBBB
 3. Old inferior infarct
 4. Consider old antero-septal infarct



9-7

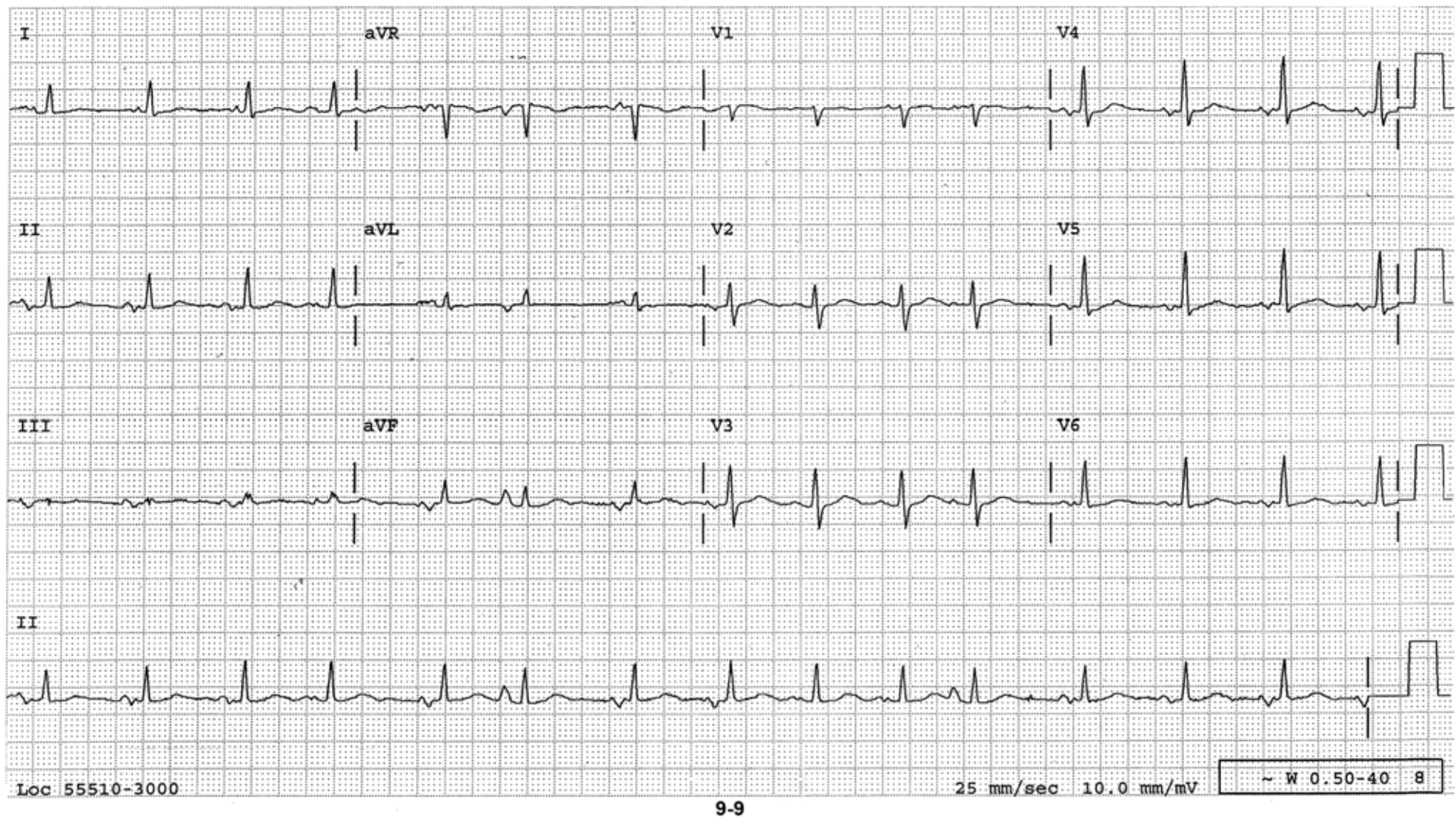
- 9-7 A narrow QRS tachycardia at a rate of 150/minute. The findings in lead V_2 is suggestive of sinus tachycardia. However, careful observation of V_1 indicates two atrial activities for each QRS, occurring regularly at a rate of about 300/minute which is diagnostic of atrial flutter. This will force one to look at the inferior leads for the "sawtooth pattern." Leads II and III, or better yet, aVF indeed reveals sawtooth pattern of atrial flutter with every other sawtooth being deformed by the QRS. Another example of atrial flutter with 2:1 AV conduction which is one of the most difficult rhythms to recognize. V_1 is useful in revealing the extra atrial activity as in this case.

Dx: Atrial flutter with 2:1 AV conduction



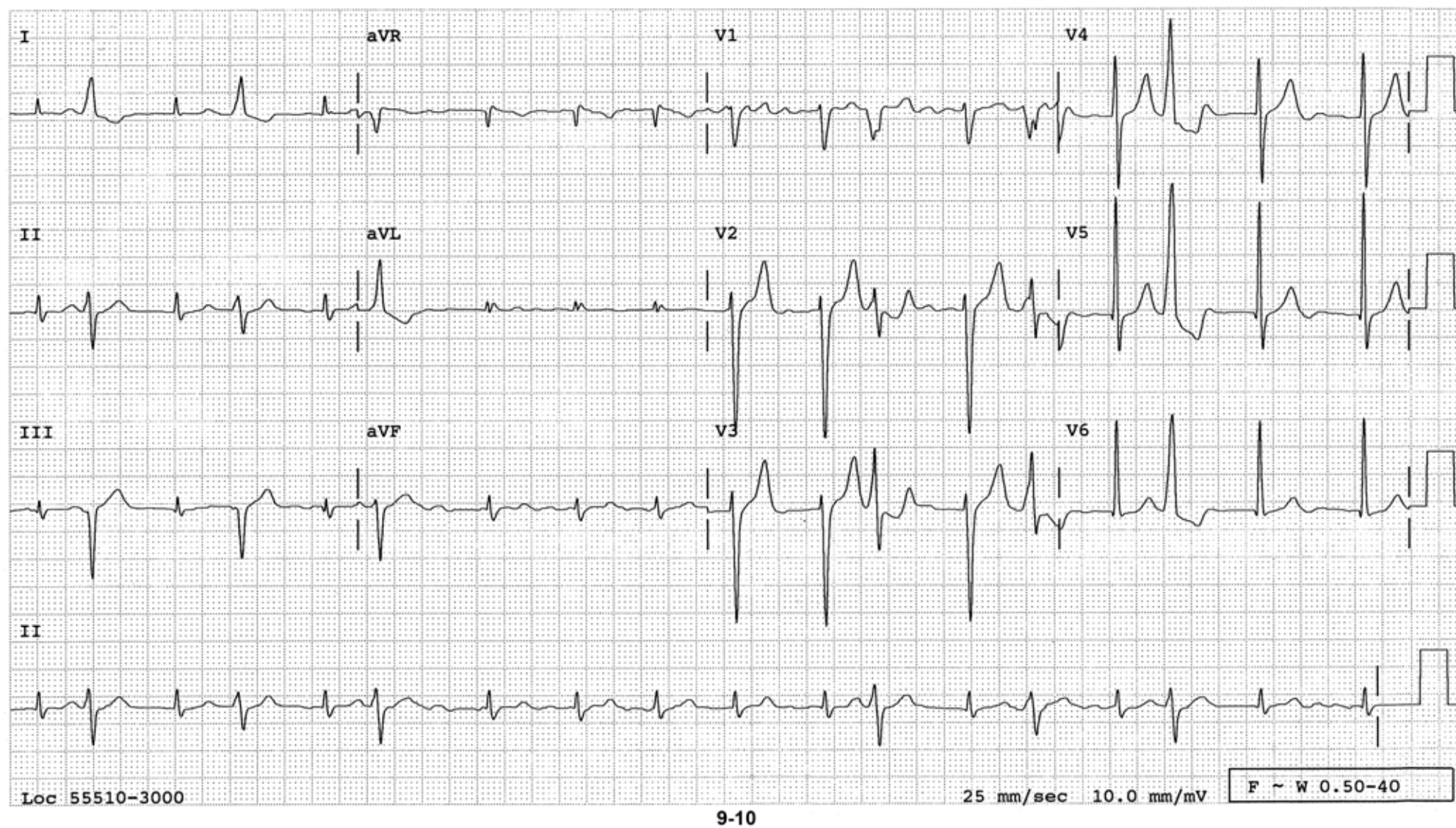
9-8 Narrow QRSs are occurring very regularly at a rate of 32/minute. Each QRS is preceded by a P wave which has a fixed P-R interval. Another P wave can be seen between the QRSs with regular P-P intervals indicating that this is not a sinus bradycardia, but sinus rhythm with 2:1 AV block. Since the QRSs are narrow and the conducted P-R interval is long, the block is within the AV node. This is a golden opportunity to appreciate how U and P waves look different. U wave has more duration to it and is a gentle mound while P wave is shorter in duration and is pointed (see leads V_2 and V_3).

Dx: Sinus rhythm with 2:1 AV block, most likely block within the AV node



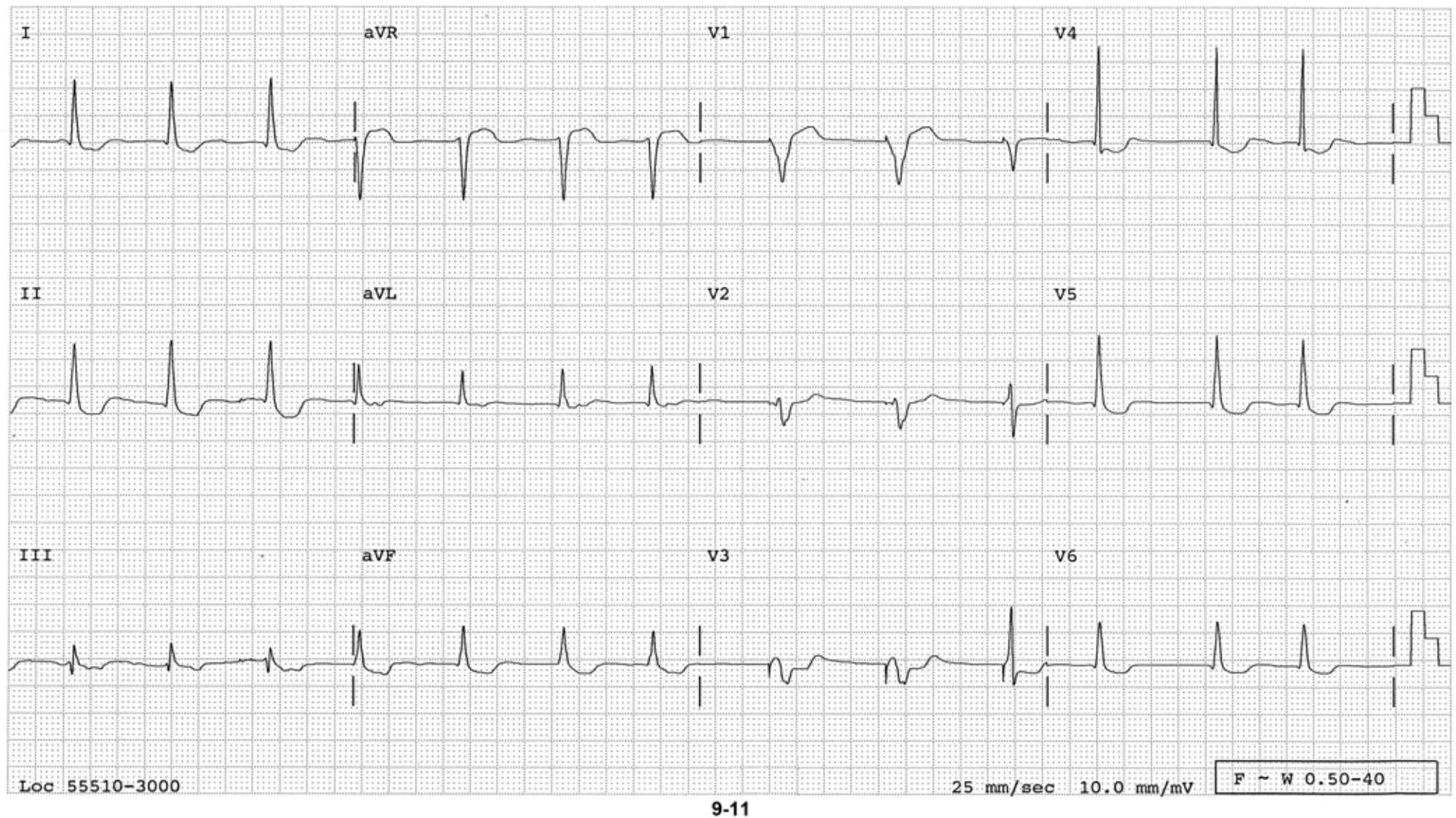
9-9 Irregularly irregular rhythm at 88/minute. QRSs are narrow. There is a discrete P wave in front of each QRS, ruling out atrial fibrillation. These P waves occur irregularly and have changing morphology. Since the rate is less than 100/minute, this is wandering atrial pacemaker, not MAT. Consider that every beat is arising from a different focus in the atria and is a benign condition.

Dx: Wandering atrial pacemaker, benign



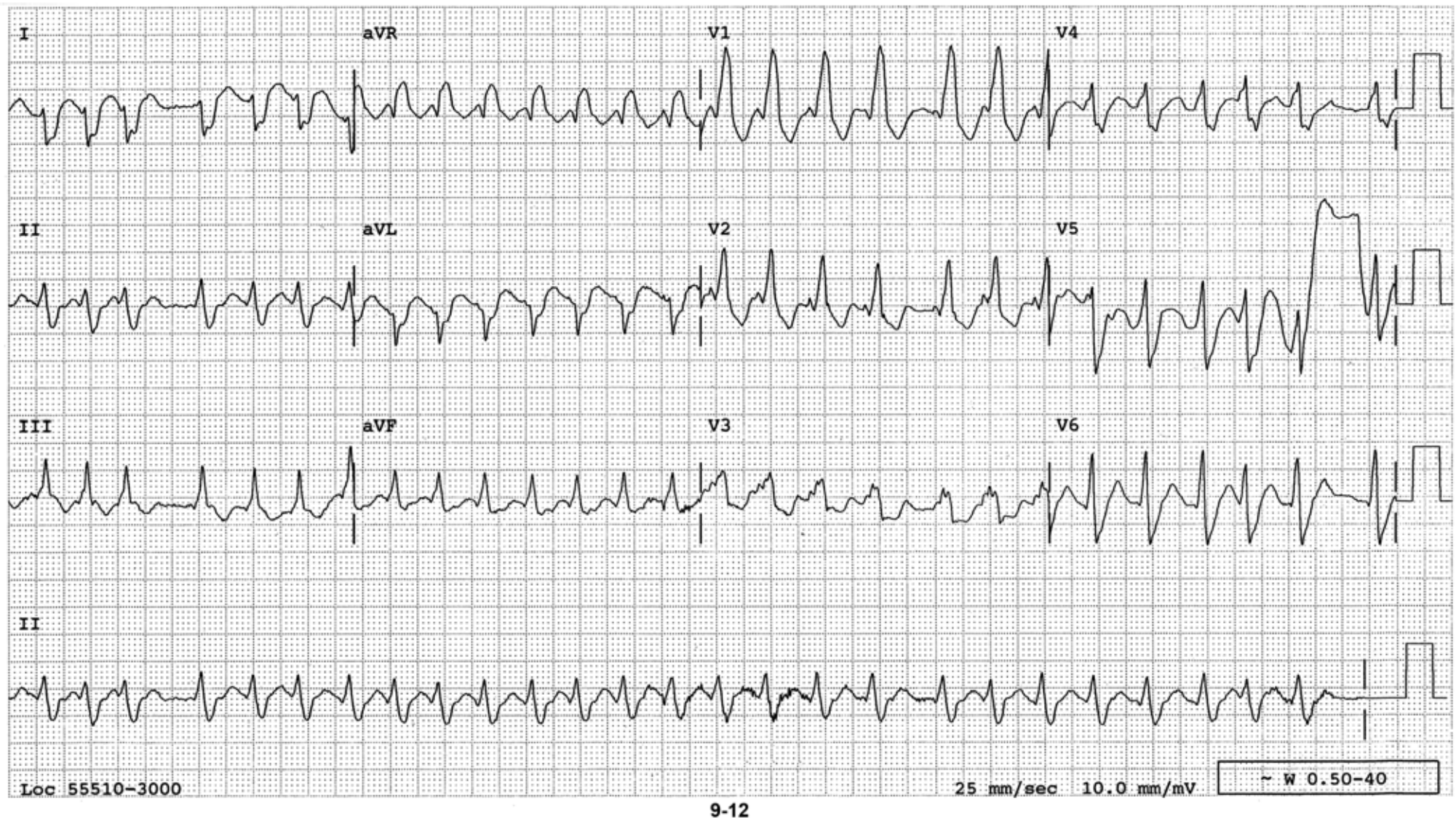
9-10 Atrial fibrillation with a ventricular rate of about 115/minute. Frequent ventricular premature complexes are noted. There is a voltage criterion for LVH.

- Dx:*
1. Atrial fibrillation
 2. PVCs
 3. LVH by voltage



9-11 Irregularly irregular rhythm at a rate of 69/minute. No distinct P waves are present indicating atrial fibrillation. When the ventricular rate slows down below the demand pacemaker rate, the artificial demand pacemaker escapes as happened when the right precordial leads are being taken. The third paced QRS looks different from the initial two because it is a fusion of paced and normally conducted QRSs. "Scooped-out" ST depression in many leads are good for ST-T changes of digitalis effect. Voltage criteria and ST-T changes for LVH are present.

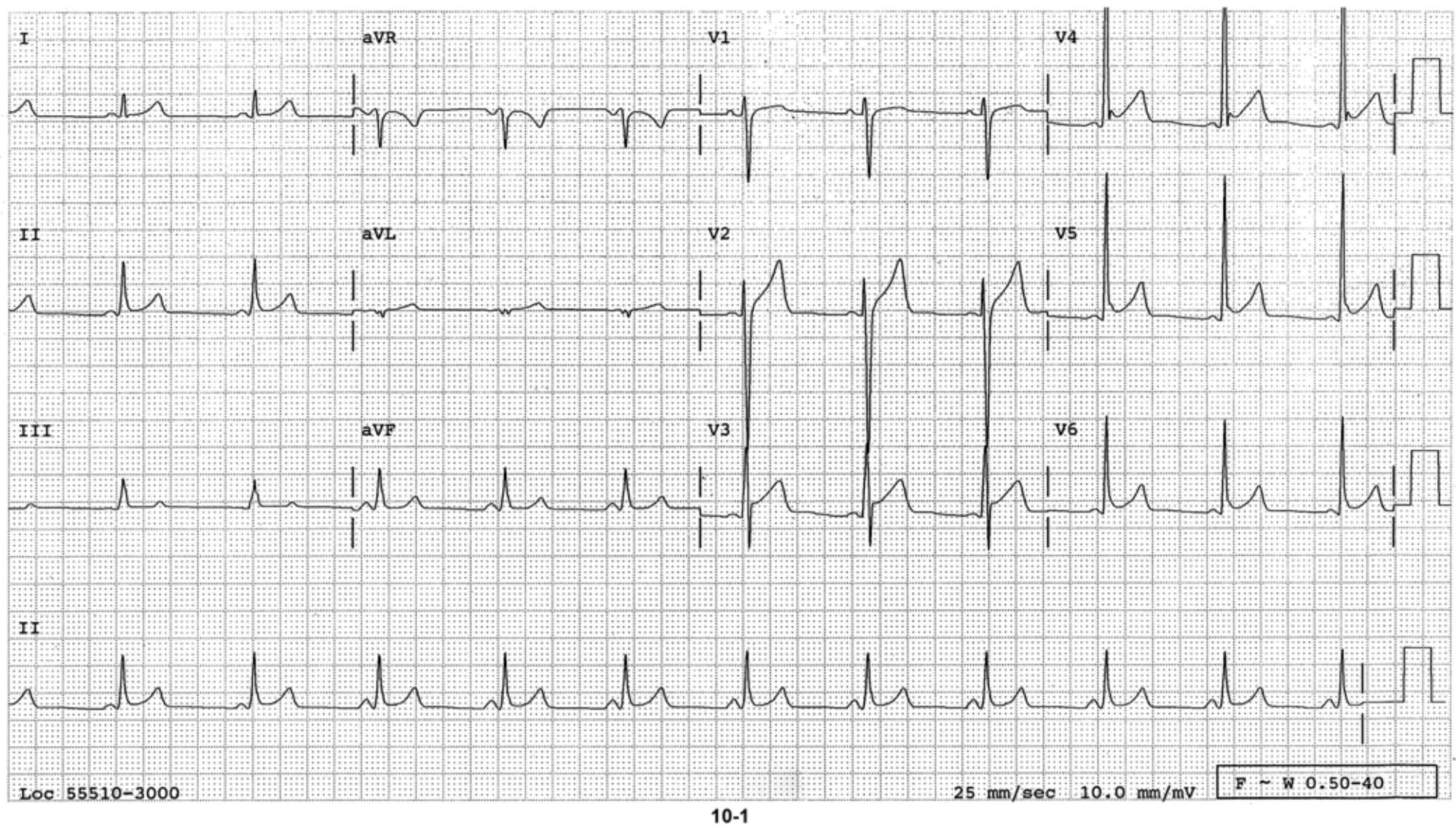
- Dx:*
1. Atrial fibrillation
 2. Appropriately functioning demand ventricular pacemaker with one fusion beat
 3. LVH
 4. ST-T changes of digitalis effect



9-12 Irregularly irregular rhythm at a rate of 166/minute with no P waves indicating atrial fibrillation. The QRSs are wide with RBBB pattern. The QRSs in lead I is mostly negative after a brief period of positivity (< 30 milliseconds) meeting the criteria of left posterior fascicular block. This is an example of BIFB consisting of complete RBBB and left posterior fascicular block.

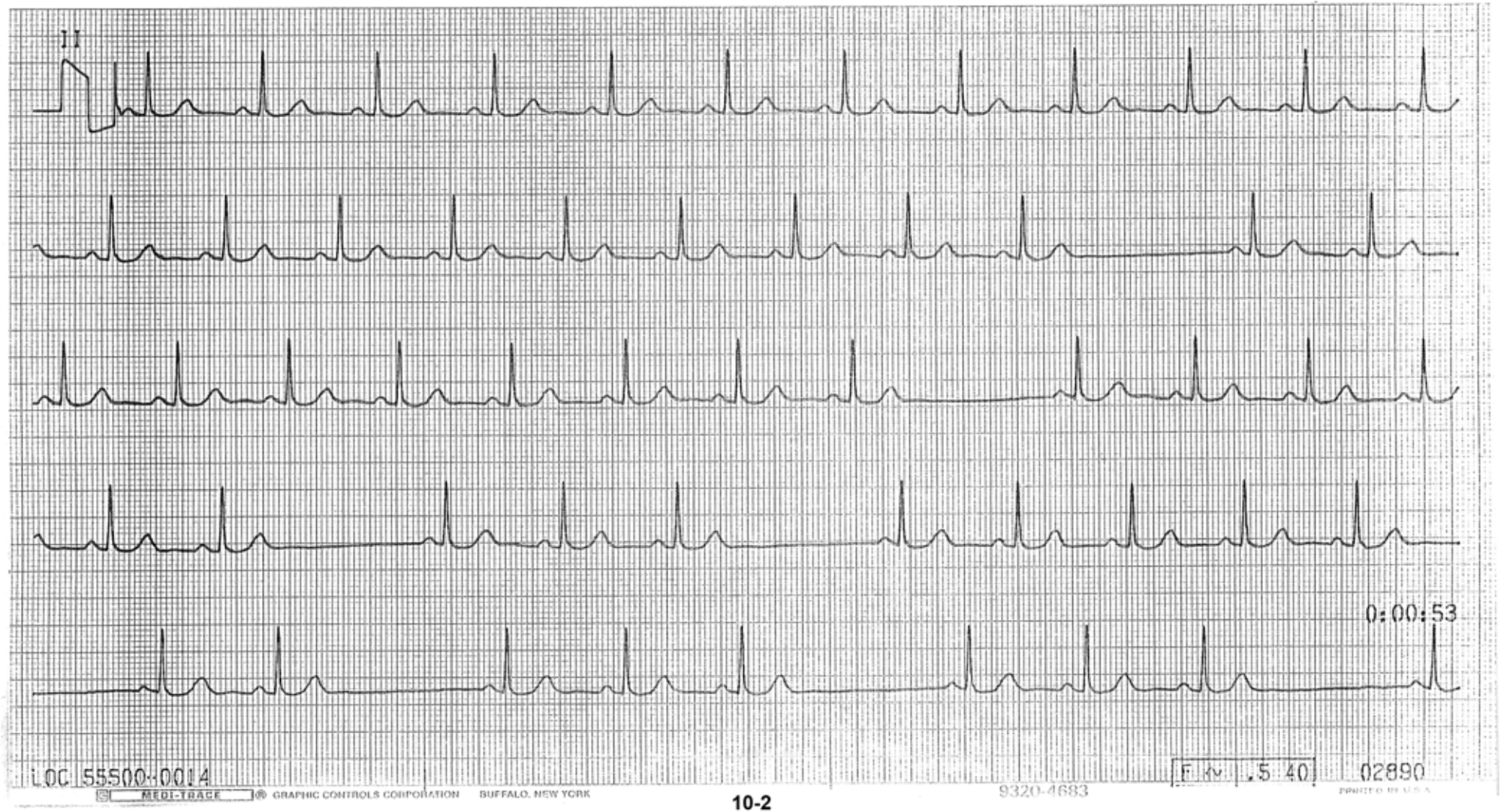
- Dx:*
1. Atrial fibrillation with a fast ventricular response of 166/minute
 2. Bifascicular block consisting of RBBB and left posterior fascicular block

SECTION 10



10-1 A regular sinus rhythm at a rate of 67/minute. The P-R interval is short measuring less than 120 milliseconds and indicates accelerated AV conduction ("greasy" AV node). There is an increased QRS voltage in the precordial leads. ST-segment is elevated in many precordial leads with concavity upward. There is a notching in the junction. These are features of early repolarization pattern as a normal variant.

- Dx:
1. Sinus rhythm
 2. Accelerated AV conduction
 3. LVH voltage
 4. Early repolarization pattern



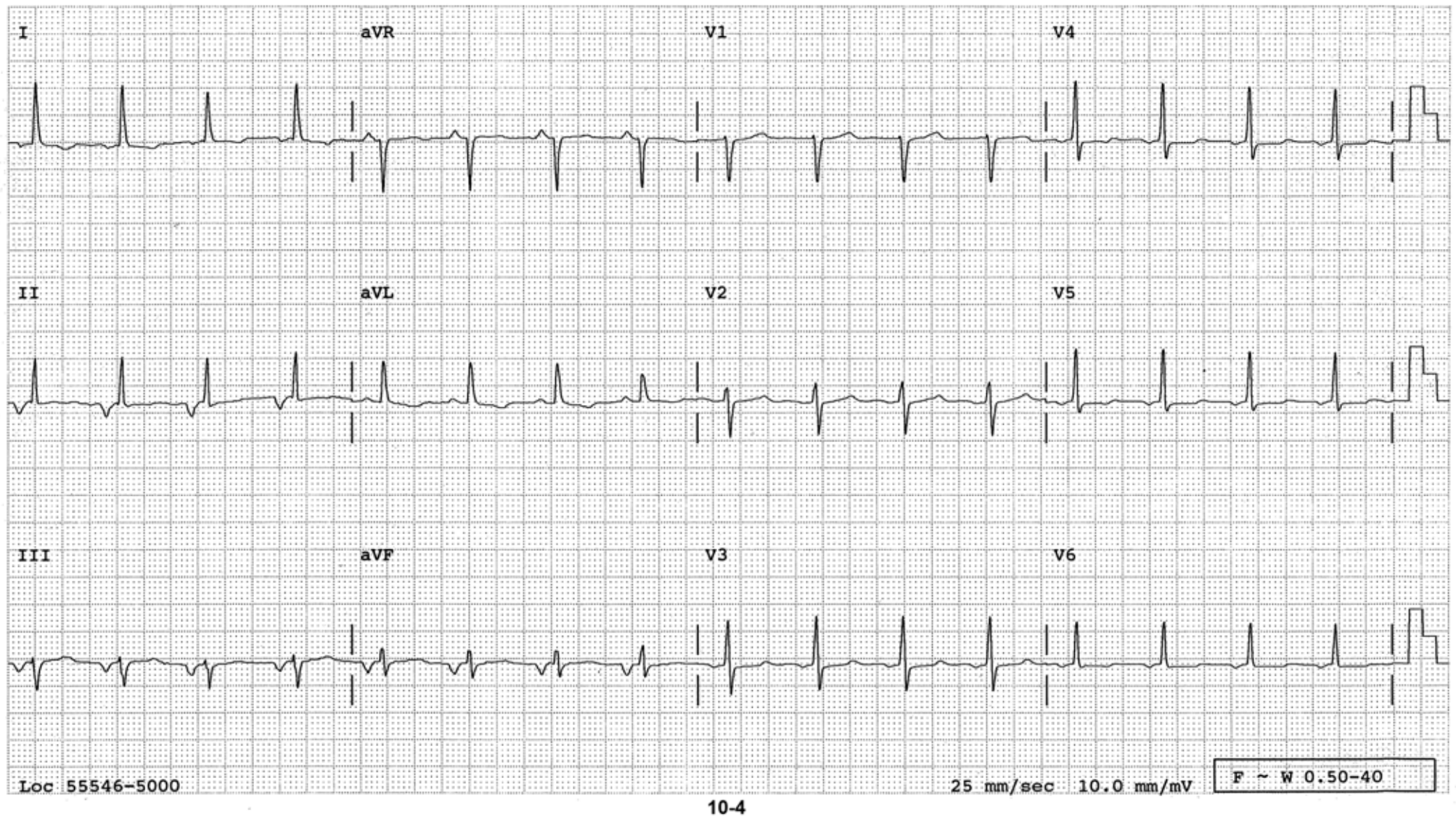
- 10-2 Regular sinus rhythm is frequently interrupted by pauses. There are no blocked P waves during the pause and the pause is exactly two times the basic cycle length, indicating sinoatrial block. This is an example of Type II SA block. SA block without any change in the R-R interval indicates that there is no progressive lengthening of the SA conduction time prior to the block, hence Type II SA block. In Type I SA block, the progressive lengthening of the SA conduction time prior to the block will result in the pause being less than two R-R intervals.

Dx: Type II SA block



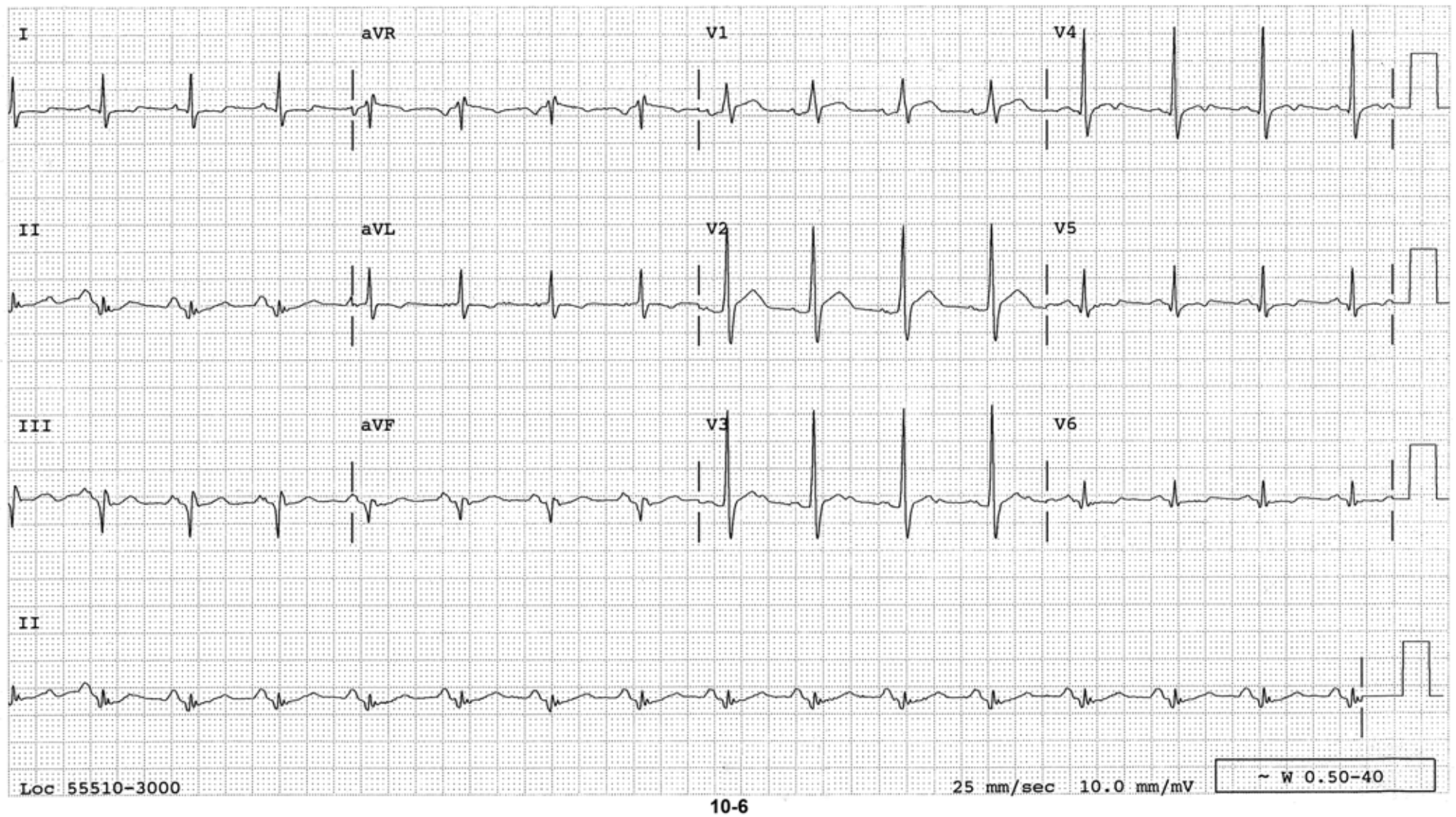
- 10-3 Sinus P waves occur regularly but at slightly slower than the AV junctional escape rate, allowing the latter to manifest (escape). Since the sinus P waves and the junctional beats occur close together during the refractory period of each other, AV dissociation results. The two QRSs marked by X occur with a shorter R-R interval than others, indicating they are induced by the sinus P waves (capture beat). The junctional rate is about 55/minute. The primary problem of this ECG manifestation is slowing of the sinus rhythm. If the sinus rate was faster or the AV junctional rate was slower, this phenomenon would not have happened. It is important to realize that there is no AV block, which is proven by the capture beats.

Dx: Sinus bradycardia causing AV junctional pacemaker to escape with AV dissociation and two capture beats



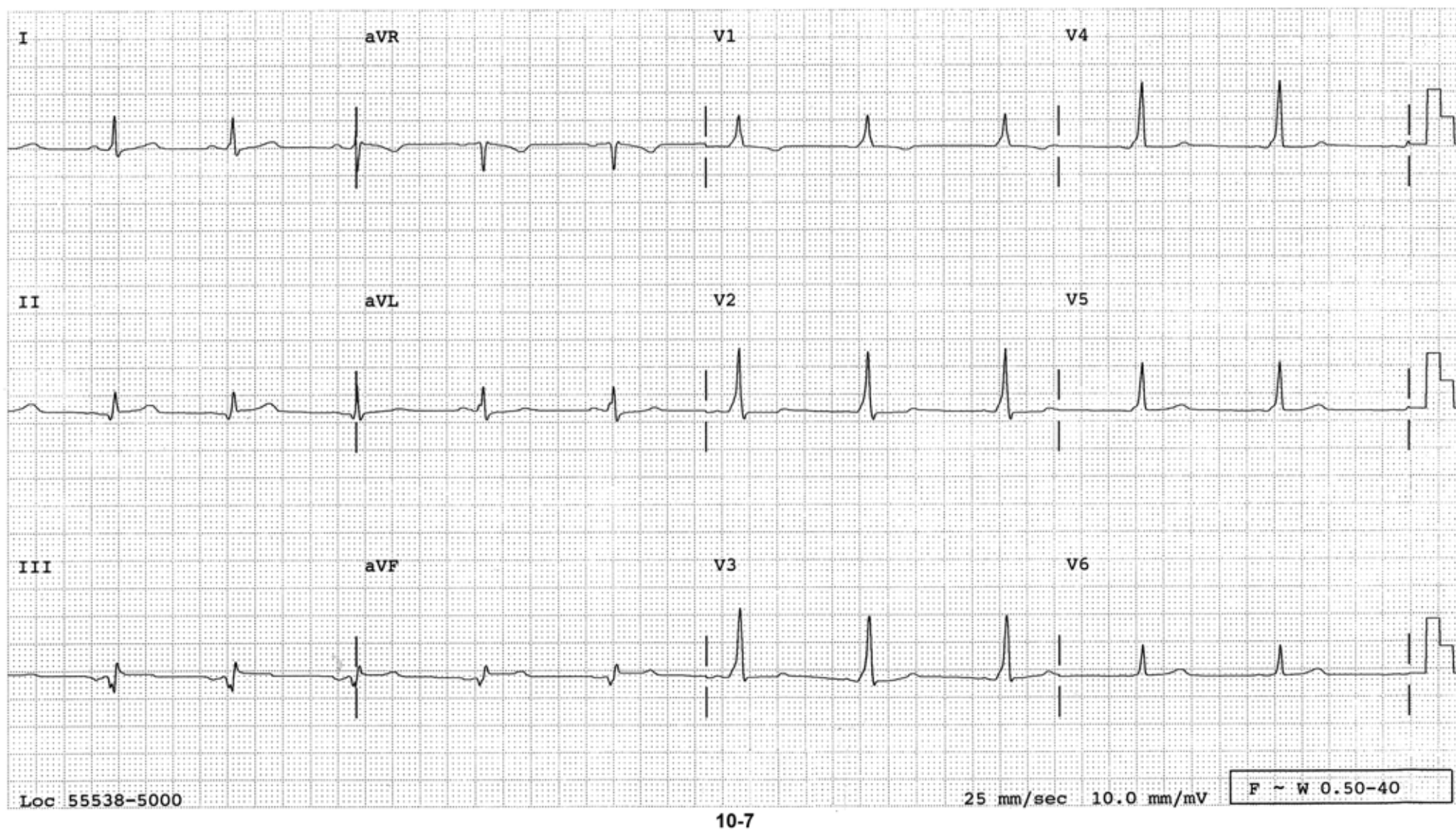
10-4 A regular rhythm at a rate of 94/minute. P waves are negative in the inferior leads indicating the atria are depolarized retrograde. This happens only if the impulse originates from the low atrium or from the AV junction. Normal P-R interval of 150 milliseconds favors low atrial rhythm while if it were shorter than that, one would have thought of junctional rhythm. Low atrial rhythm is benign. Voltage criteria and ST-T changes for LVH are present.

- Dx: 1. Low atrial rhythm, benign
2. LVH



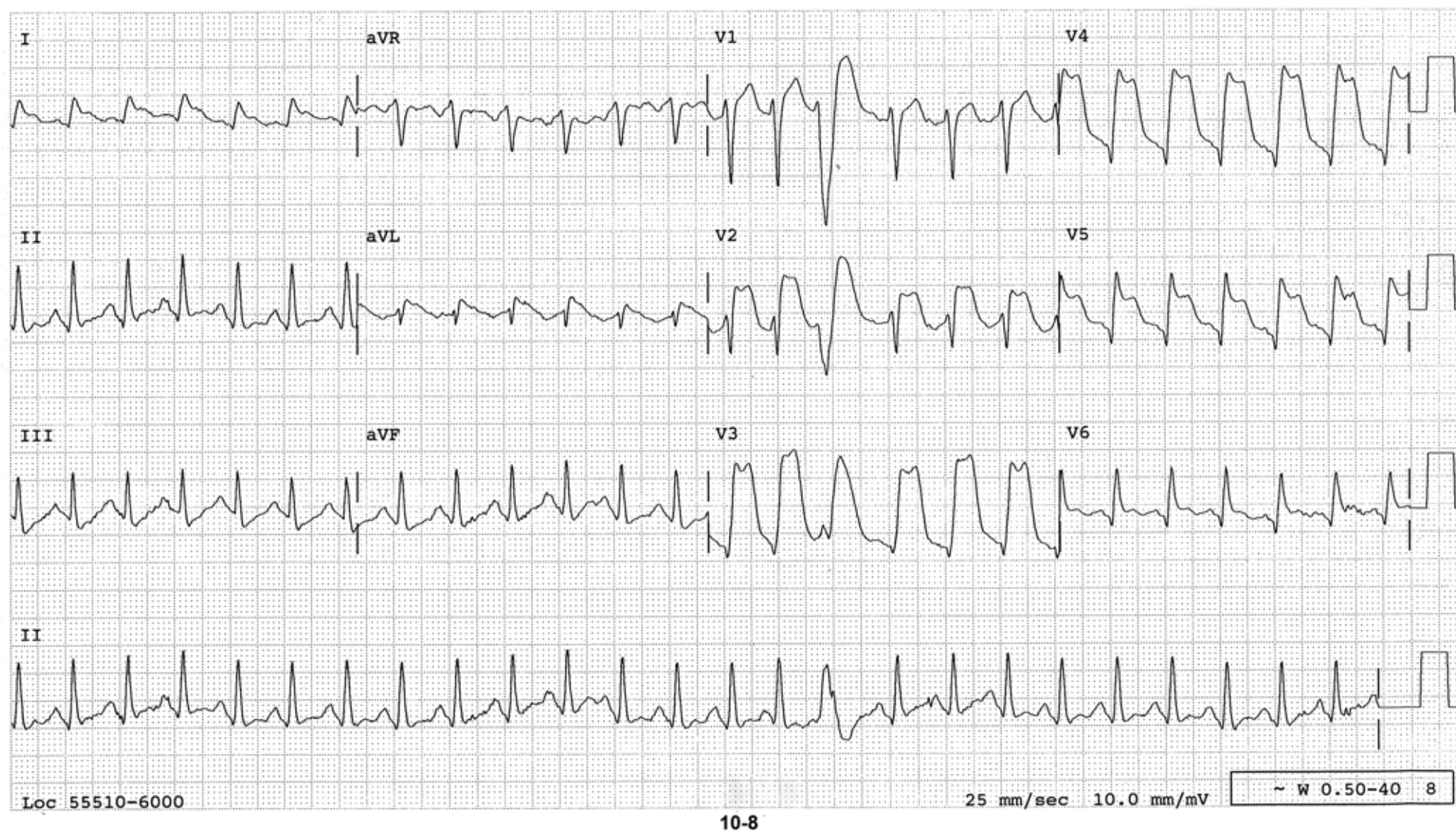
10-6 Normal sinus rhythm at a rate of 92/minute. The R waves are tall in the right precordial leads. In this case, there is an old inferior infarction and the tall R waves in the right precordial leads are due to the posterior wall being involved in the infarction process.

- Dx: 1. NSR
2. Old inferoposterior myocardial infarct



10-7 Normal sinus rhythm at a rate of 64/minute. Short P-R interval with delta waves in the precordial leads is diagnostic of WPW syndrome. The P-R interval is normal in lead I, aVL and aVR indicating the delta waves are mostly isoelectric in these leads. The negative delta waves in the inferior leads and tall R waves in V₁-V₃ simulate inferoposterior MI.

- Dx:
1. NSR
 2. WPW syndrome simulating inferoposterior infarct



10-8 Sinus tachycardia at a rate of 151/minute. Leads V_3 and V_4 suggest wide QRS tachycardia but, judging from other leads, the ST-segment is elevated to the top of the R wave, making it look like a wide QRS. This is an example of extensive anterior MI involving all precordial leads and leads I and aVL. Leads I and aVL represent high lateral left ventricular wall, which is ordinarily perfused by a diagonal branch, which takes off LAD proximally. Therefore, the ECG findings in this case indicate proximal LAD occlusion. If only the precordial leads are involved, the culprit lesion is in the LAD not proximal. If only leads I and aVL are involved without the precordial leads, the lesion is in the diagonal branch. Thus, ECG findings will often tell us where the culprit lesion is. One PVC is present in the middle of the strip.

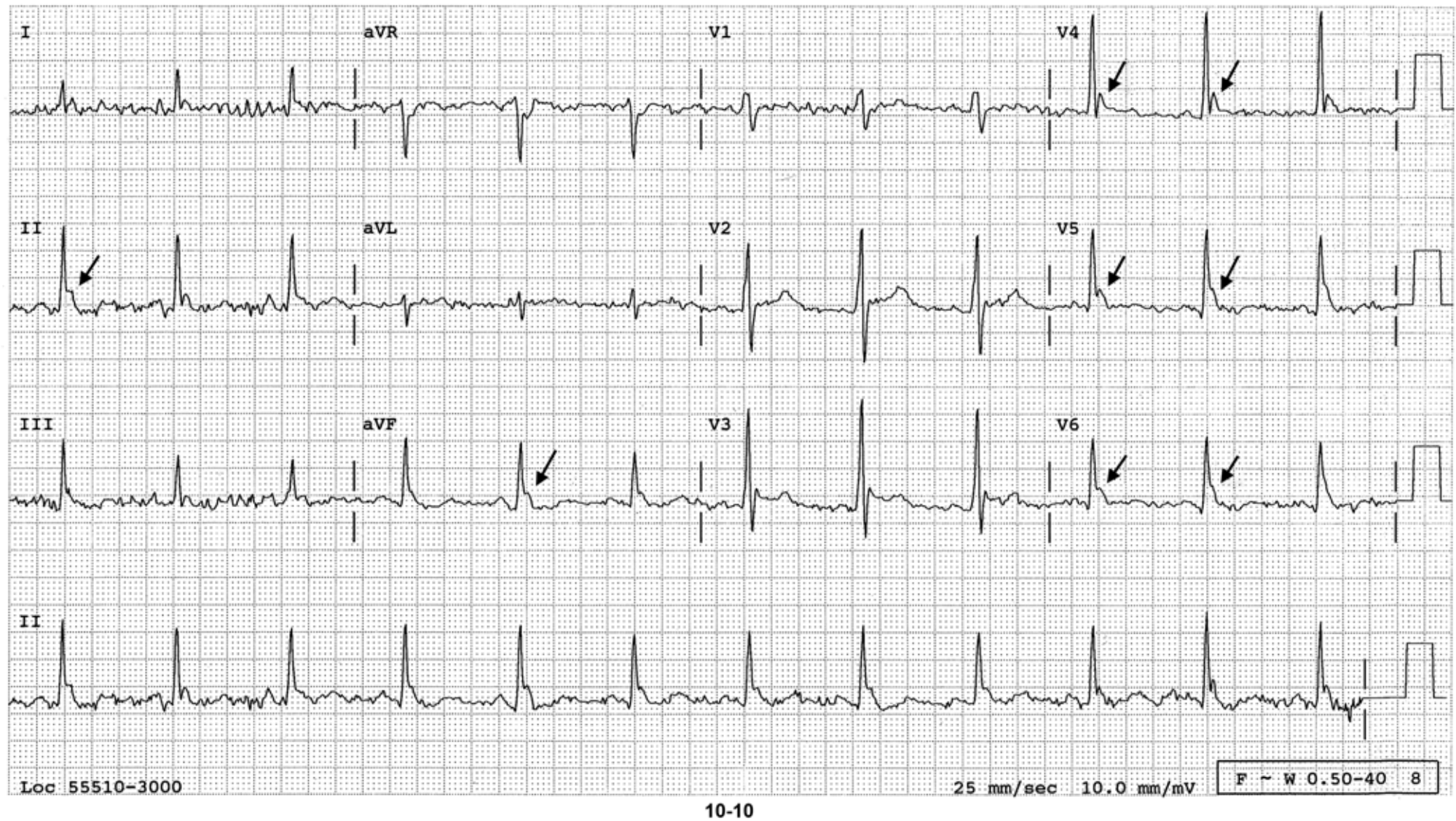
- Dx:*
1. Sinus tachycardia
 2. PVC
 3. Extensive anterior STEMI



10-9

10-9 A regular rhythm at a rate of 90/minute. The QRSs are narrow. There is no P wave in front of the QRS. However, there is a negative blip following the QRS in the inferior leads indicating AV junctional rhythm with 1:1 retrograde conduction to the atria. The junctional rate is not faster than 130/minute, but is faster than its intrinsic junctional rate and this will be called accelerated AV junctional rhythm.

Dx: Accelerated AV junctional rhythm with 1:1 retrograde conduction to the atria



10-10 Sinus rhythm at a rate of 71/minute. The QRSs are wide. The most striking feature of this tracing is a slurred down stroke (↓), an opposite from a delta wave, especially noticeable in the left precordial leads. This is called either J wave or Osborn wave and is diagnostic of hypothermia. The baseline is not stable due to the patient shivering.

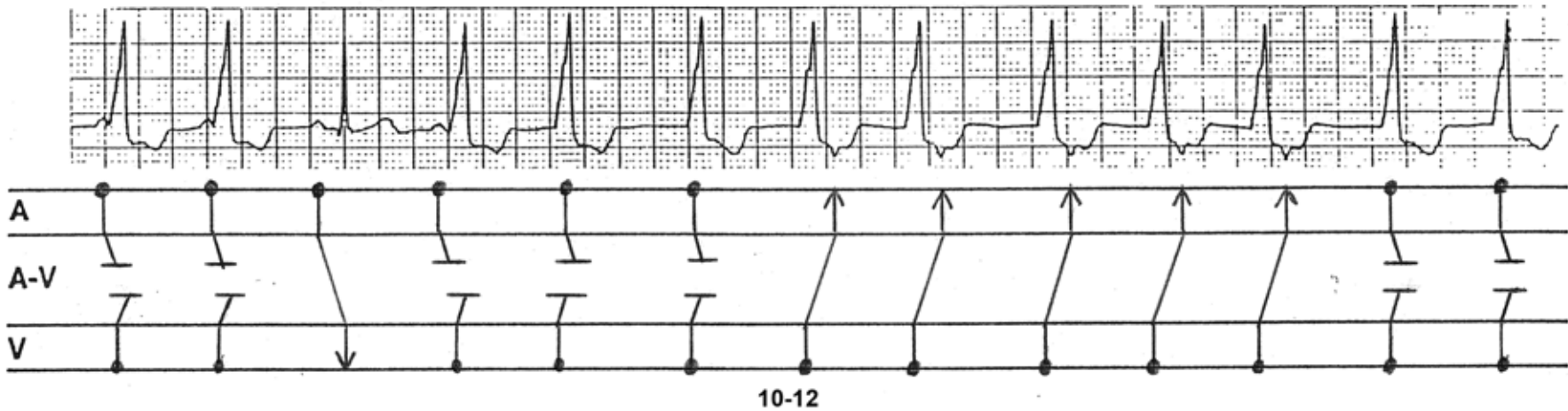
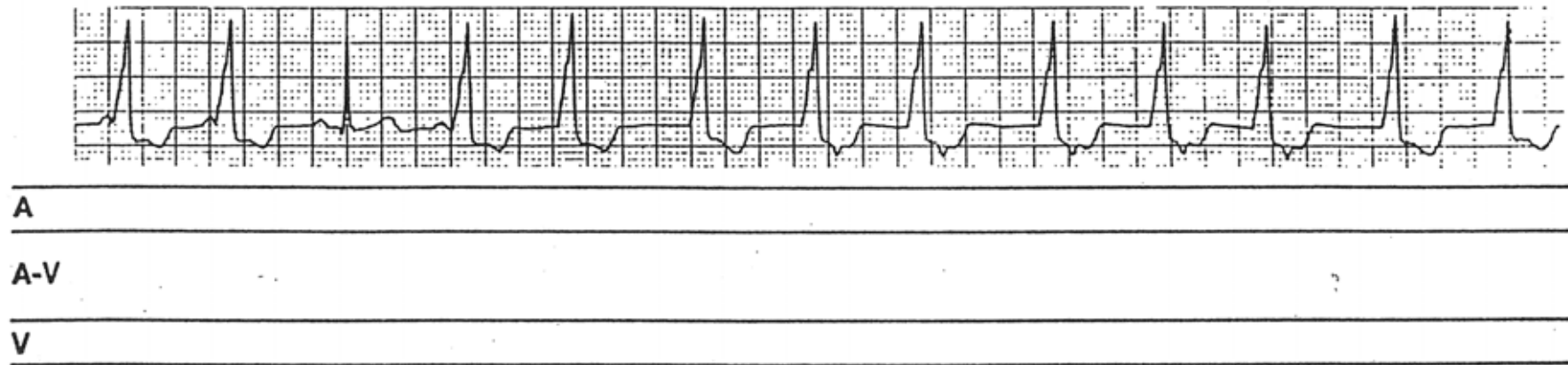
- Dx:
1. NSR
 2. Osborn wave from hypothermia
 3. Artifact in the baseline from shivering



10-11 Sinus tachycardia at a rate of 122/minute. RBBB is present. P wave is very tall measuring 5 mm in lead II indicating RAE. The RBBB in this patient may be due to RVH. The P wave in V_1 is mostly negative indicating LAE as well.

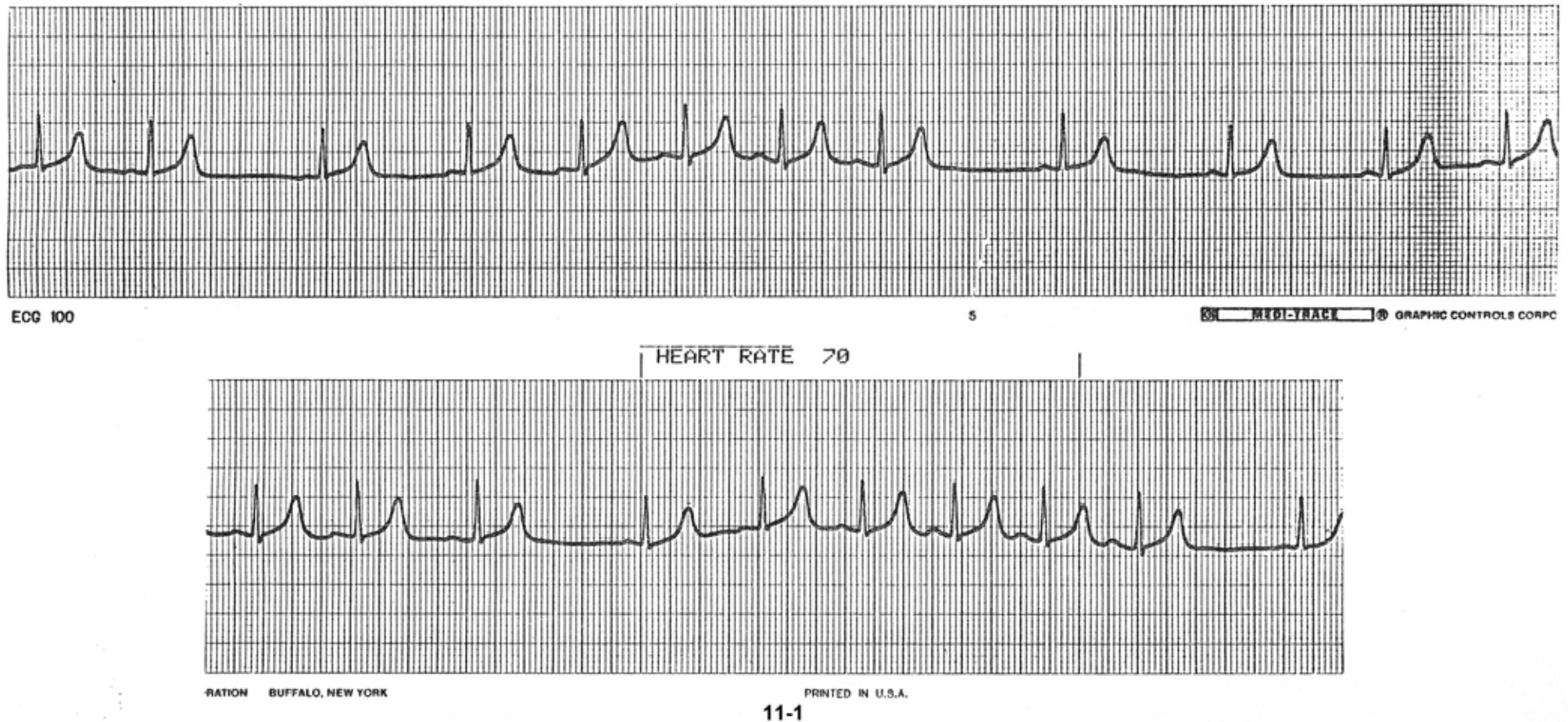
- Dx:*
1. Sinus tachycardia
 2. RBBB
 3. Biatrial enlargement
 4. Consider RVH

Please draw the ladder diagram in the space provided below.



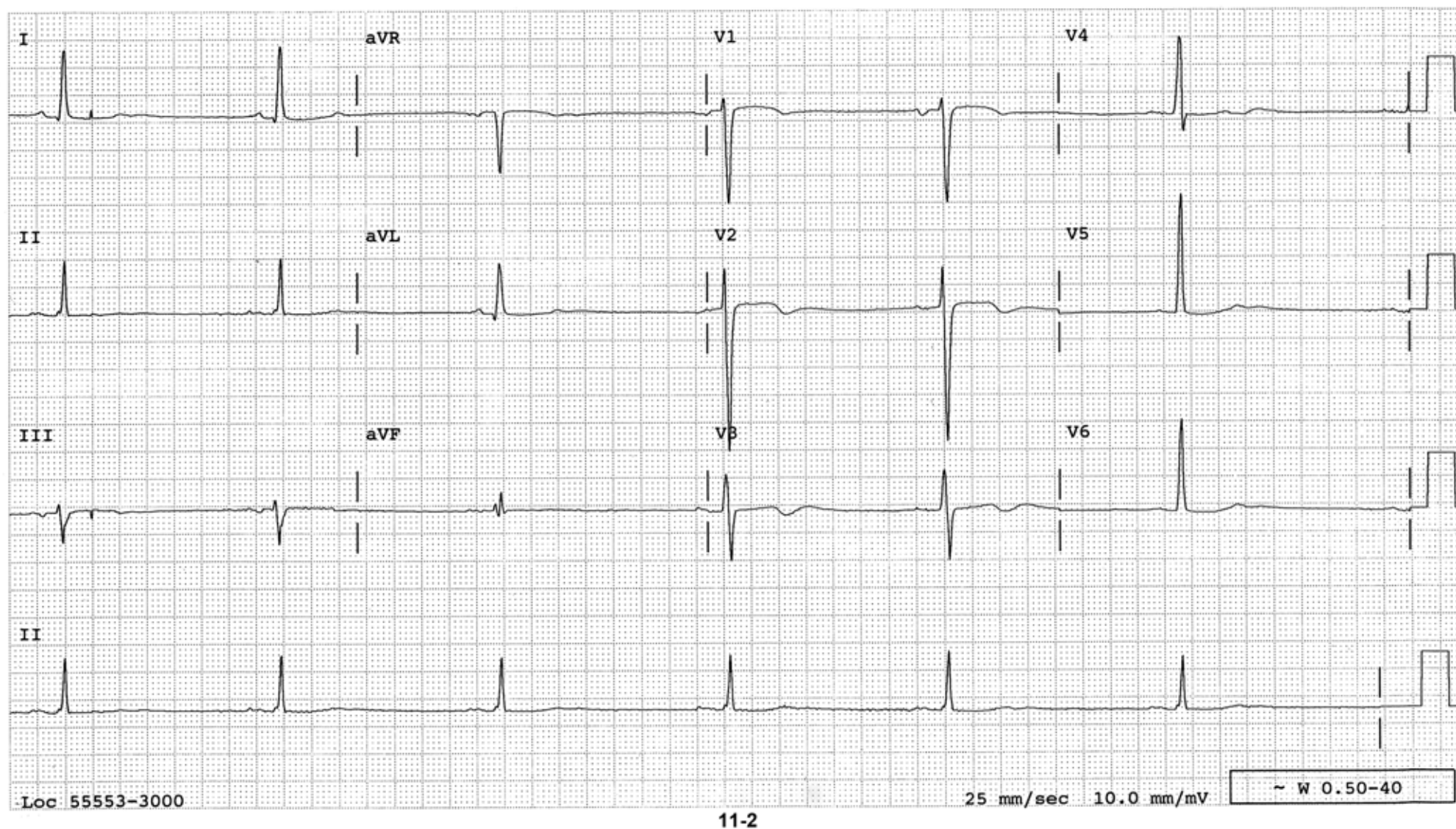
10-12 This is an example of an accelerated idioventricular rhythm with AV dissociation at the beginning, then 1:1 retrograde conduction to the atria and AV dissociation again as diagrammed. One capture beat (third QRS) is present.

SECTION 11



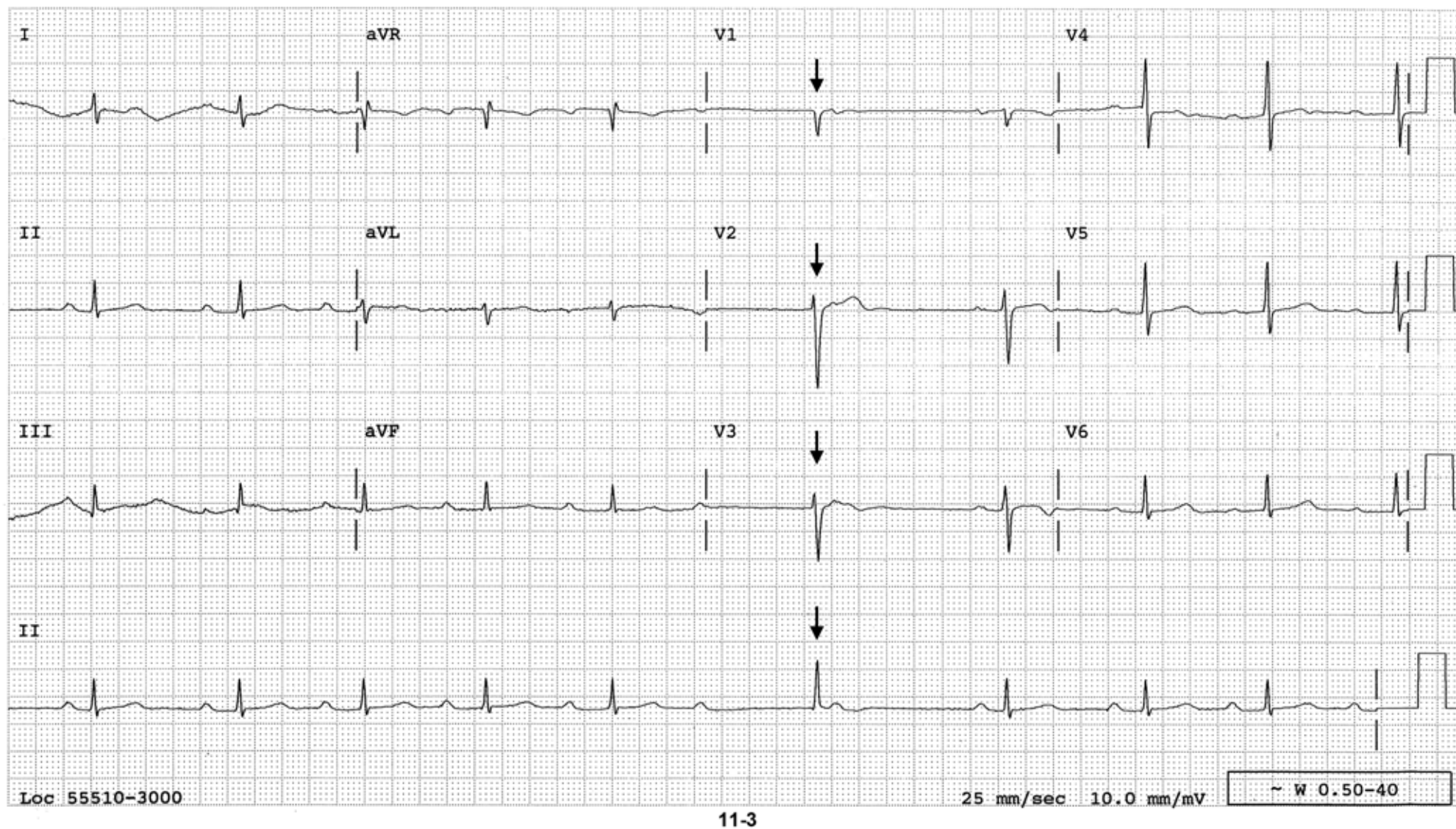
- 11-1 An irregular rhythm is present. Every QRS is preceded by a P wave with a fixed P-R interval indicating a sinus rhythm. The rate gradually speeds up and then gradually slows down in a rhythmic fashion. This is a good example of sinus arrhythmia, that is, the HR fluctuation related to respiration. During inspiration, the HR speeds up, and during expiration, the HR slows down. The keyword for sinus arrhythmia is gradual. The rate speeds up gradually and slows down gradually. The younger the patient, the more the sinus arrhythmia.

Dx: Sinus arrhythmia



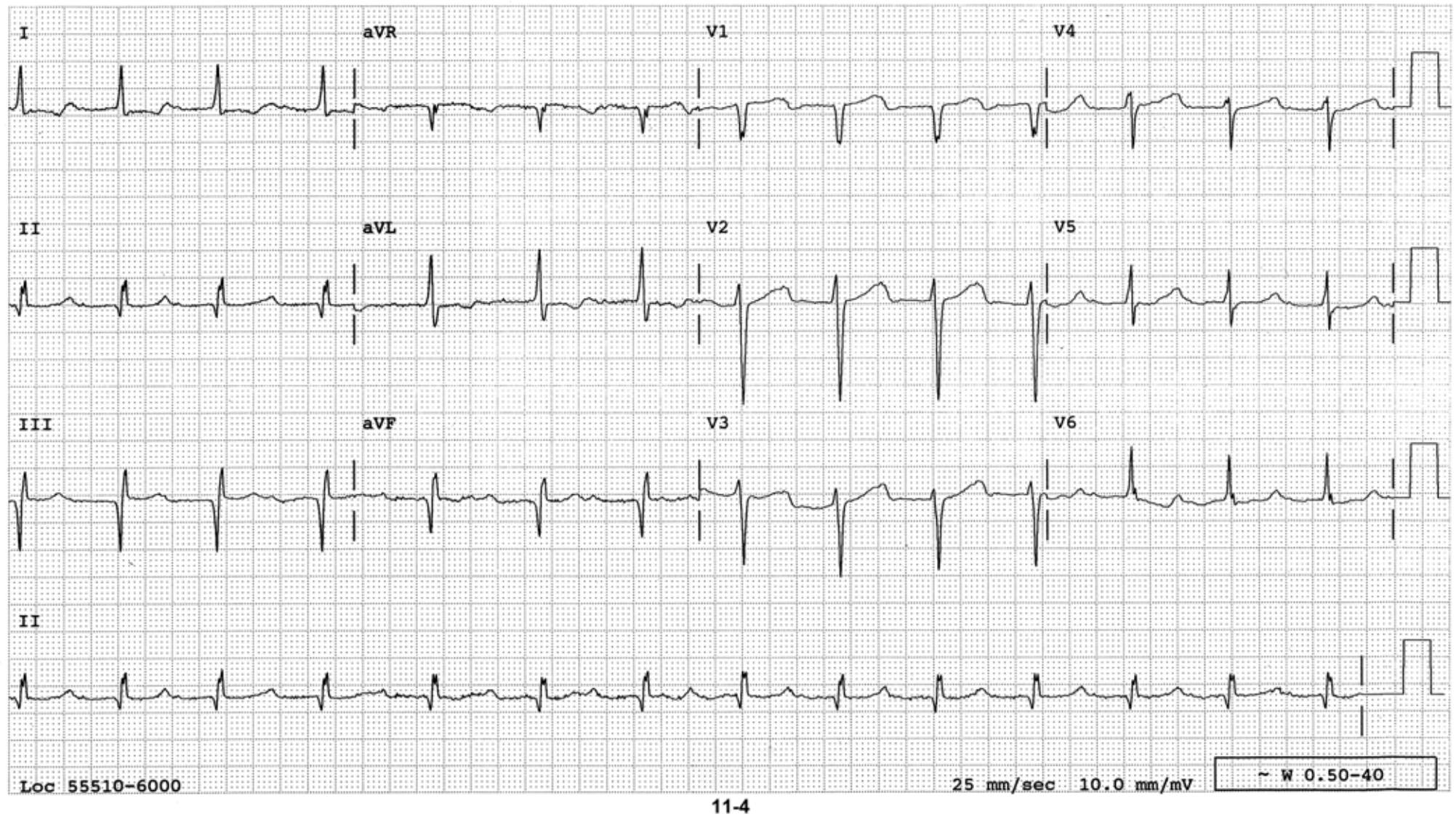
11-2 A regular rhythm at a rate of 37/minute. The QRSs are narrow and each of them is preceded by a P wave. There is no additional P wave between the QRSs indicating sinus bradycardia rather than a sinus rhythm with 2:1 AV block, or nonconducted PACs causing the slow rhythm. LAE and voltage criteria and mild ST-T changes for LVH are also present.

- Dx:*
1. Sinus bradycardia
 2. LAE
 3. Probable LVH



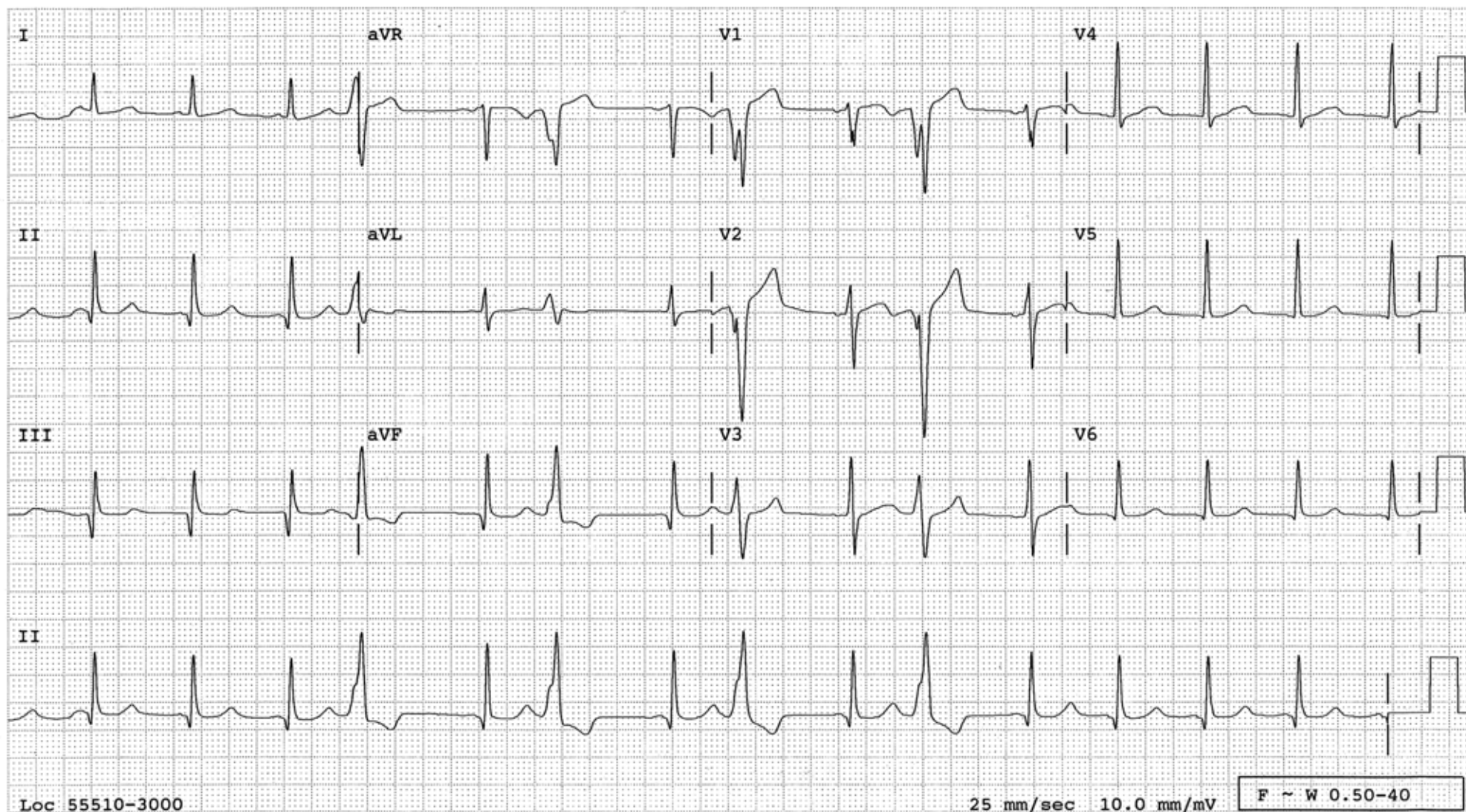
11-3 A typical AV Wenckebach phenomenon is present. In the middle of the strip, there is a junctional escape complex after the blocked P wave (↓). Ordinarily, the P wave after the blocked P wave is the beginning of the next Wenckebach cycle. However, if the pause is longer than the junctional escape interval, junctional beat will escape.

- Dx: 1. Type I 2° AV block
2. One junctional escape complex



11-4 Sinus rhythm at a rate of 81/minute. The P wave occurs near the end of the T wave (best appreciated in lead aVF) with a prolonged P-R interval, indicating 1° AV block. The Q waves in the inferior leads are deep and wide indicating old inferior MI. The Q-T interval is prolonged to 480 milliseconds.

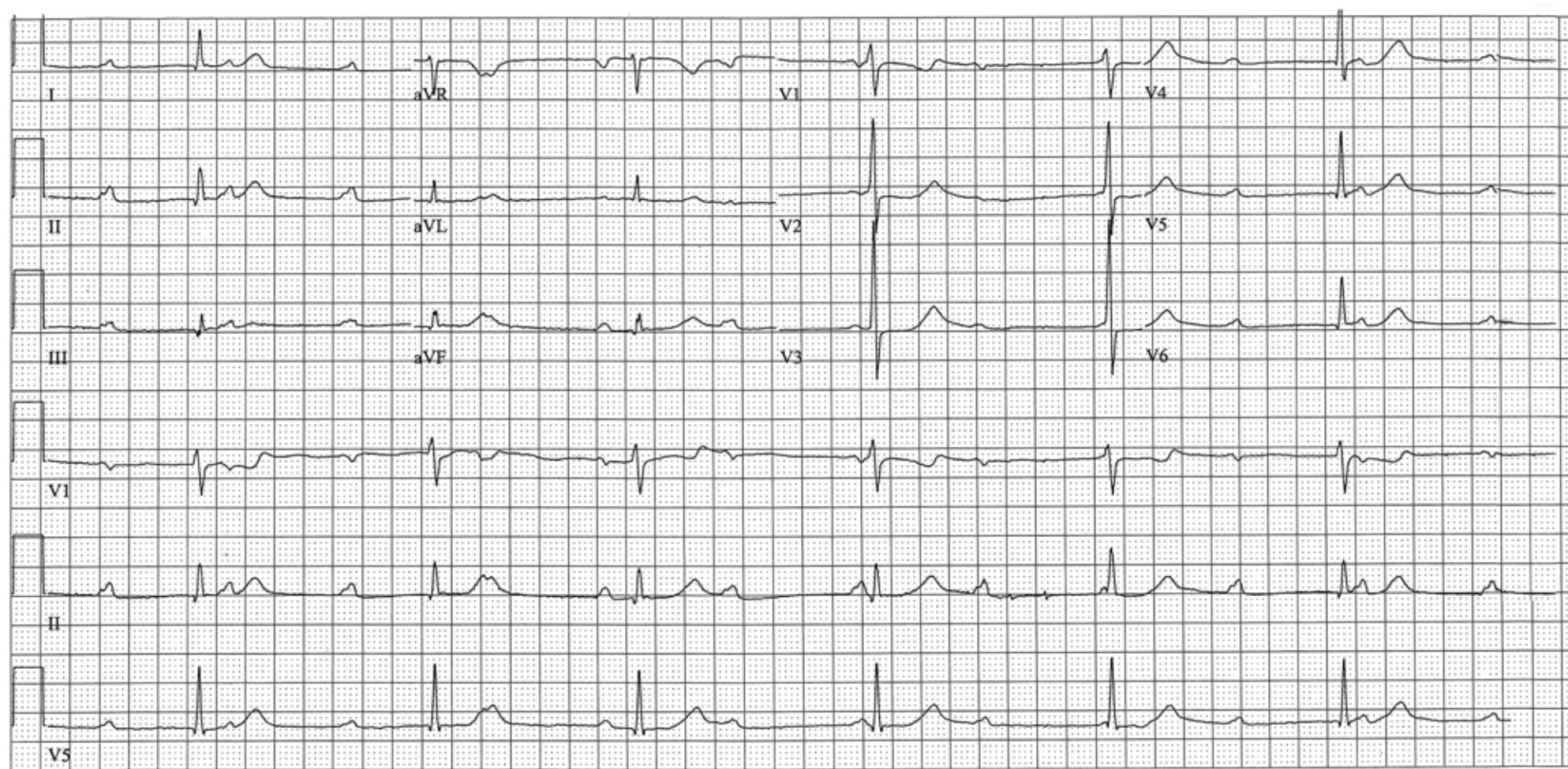
- Dx:*
1. Sinus rhythm
 2. 1° AV block
 3. Long Q-T interval
 4. Old inferior infarct



11-5

11-5 Normal sinus rhythm at a rate of 80/minute. P-R interval is short (~100 milliseconds) but there are no delta waves. This finding is considered normal, and is called accelerated AV conduction (greasy AV node). Frequent PVCs are present. The Q waves are deep and wide in lead III indicating old inferior MI.

- Dx:
1. NSR
 2. Frequent PVCs in bigeminy
 3. Old inferior infarct
 4. Accelerated AV conduction



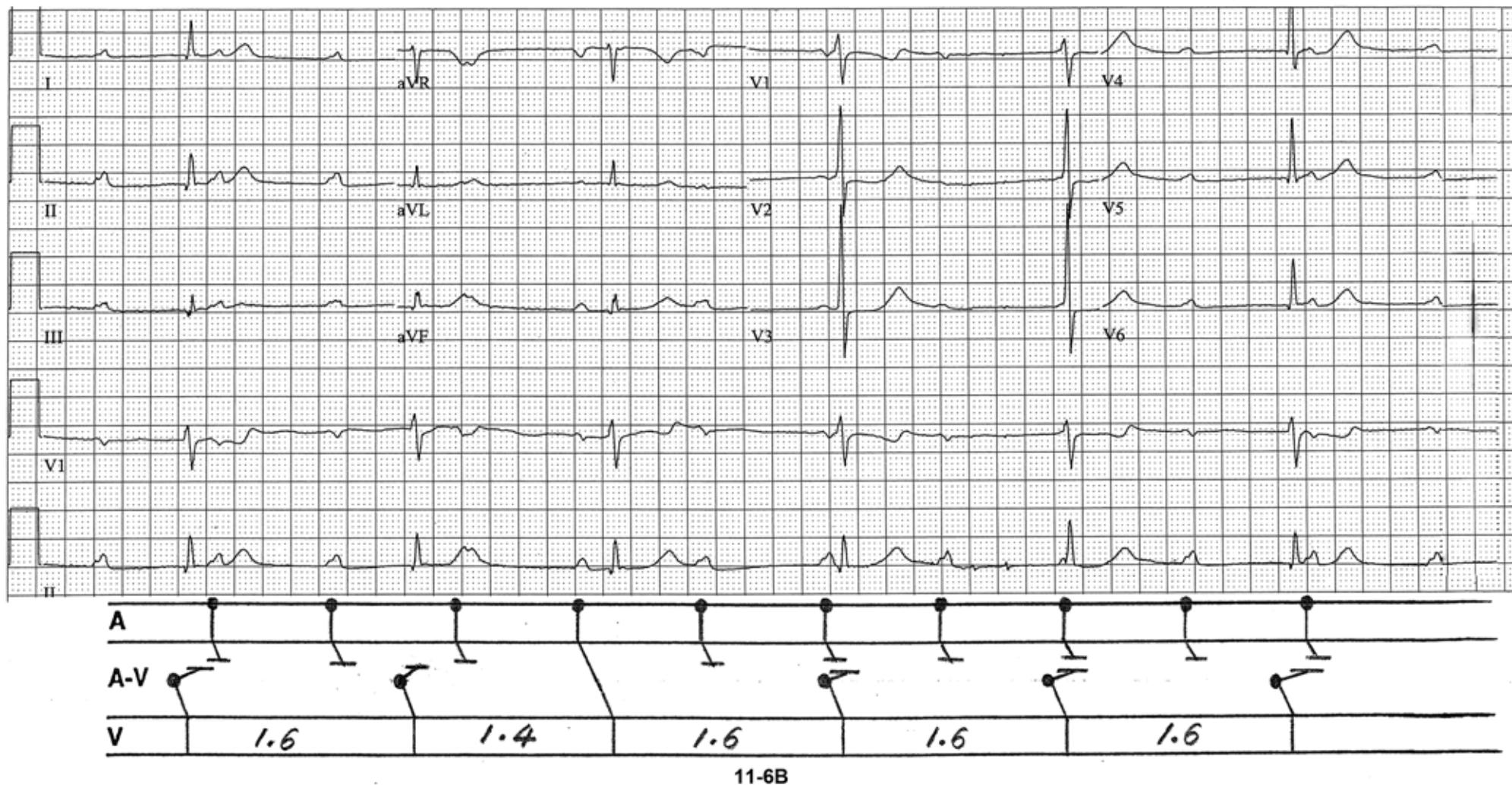
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11-6A

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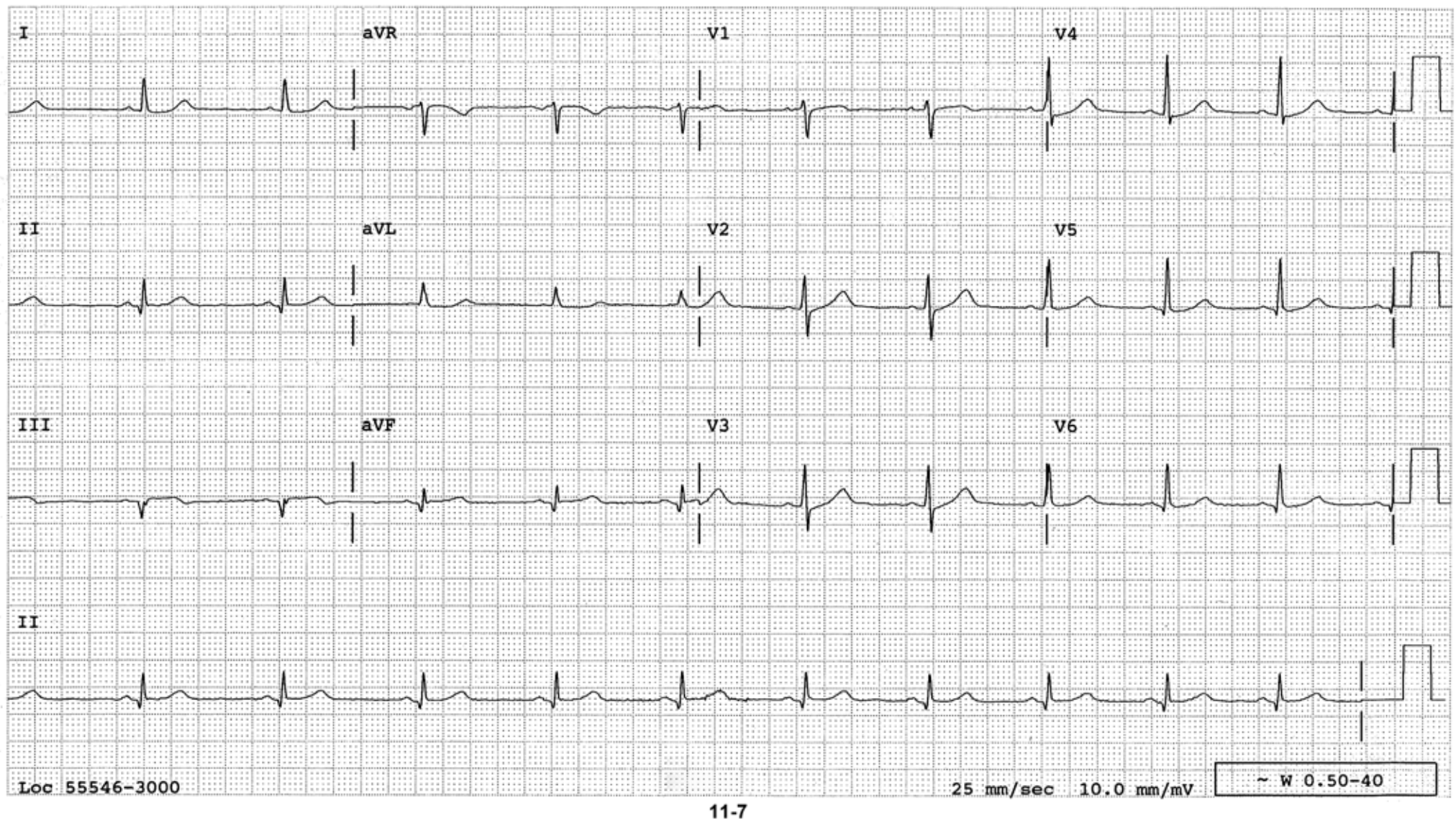
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Page 1 of 1



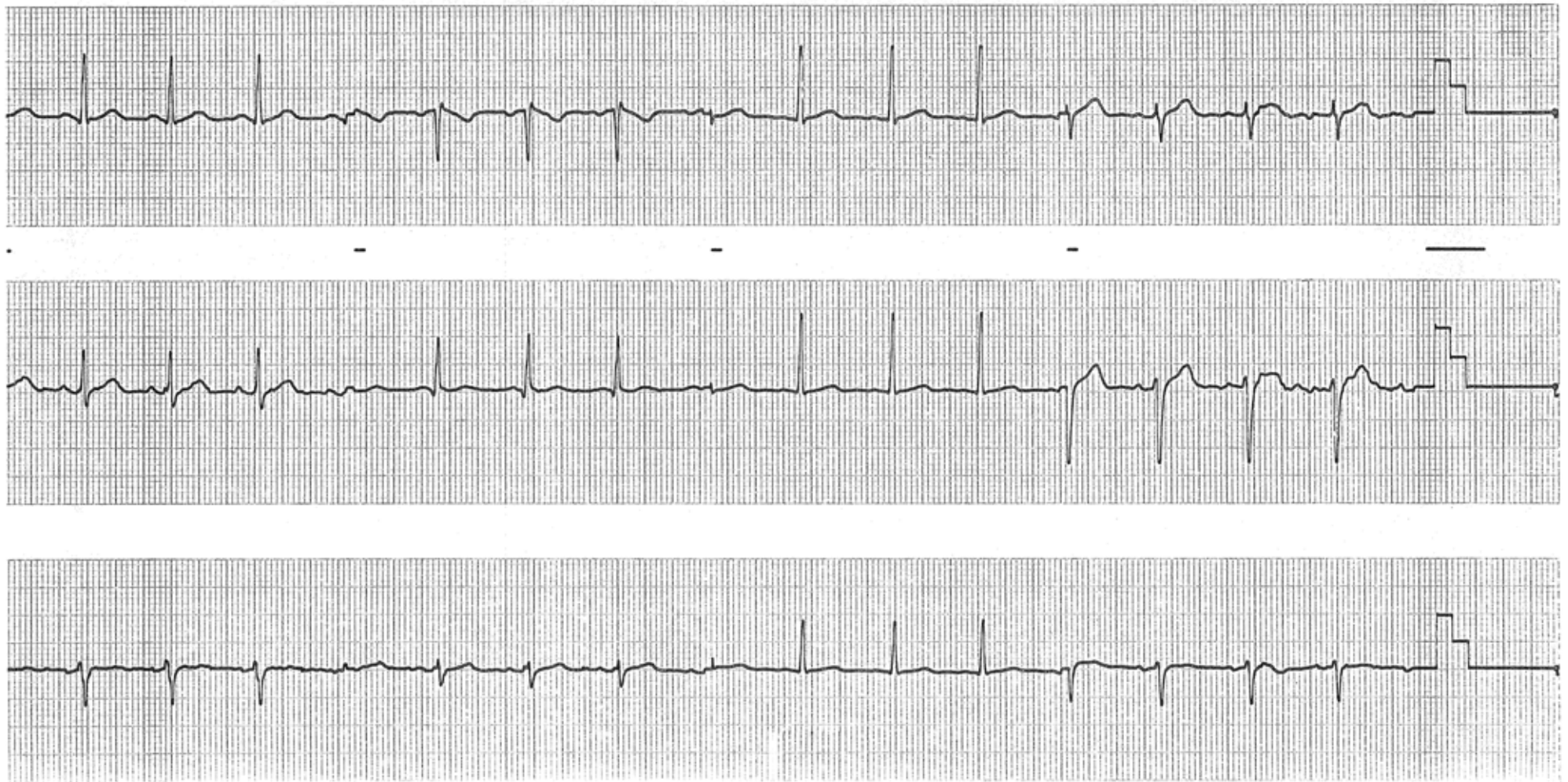
- 11-6 Narrow QRSs occur slowly at 37/minute. P waves occur regularly at 74/minute with no fixed relationship to the QRS suggesting complete (3°) AV block with AV junctional escape rhythm. The third QRS has a P wave in front of it and appears to be conducted from that P wave. This question, which is important, can be settled by measuring the R-R intervals, realizing that an AV junctional rhythm is a very regular rhythm. The third QRS occurs 1.4 seconds after the preceding QRS while all other R-R intervals are 1.6 seconds. Two hundred millisecond difference in R-R interval is sufficient and conclusive evidence that the third QRS is not a junctional beat but conducted from the P wave, proving that this patient does not have 3° AV block. What this patient has is 2:1 AV block. In a patient with 2:1 AV block, if two P-P intervals are longer than the junctional escape interval, the latter will escape. That is what is happening in this tracing. The third QRS is conducted. The next P wave is blocked because the junction cannot conduct at that interval in this patient (2:1 AV block). The next P wave was going to be conducted (how do I know? This patient's AV junction can conduct at 1.4 seconds as demonstrated by the third QRS), but the two P-P intervals (1.74 seconds) are longer than the junctional escape interval (1.6 seconds) and the latter manifests. It is important to realize that whether the next P wave will be conducted or not is determined not by the number of P waves between the QRSs but by the distance from the preceding QRS. Thus, during the shortest conductible interval, if four P waves occur, it will become 4:1 AV conduction or if only one P occurs, it will become 1:1 conduction. The junctional escape beats occur close to sinus P waves during each other's refractory period and AV dissociation results with those beats. Small Q wave in lead III and tall R waves in V_2 - V_3 suggest old inferoposterior MI.

Dx: 1. Sinus rhythm with 2:1 AV block and AV junctional escape beats with AV dissociation
2. Consider old inferoposterior MI



11-7 Normal sinus rhythm at a rate of 65/minute. QS pattern in lead III with a slight ST elevation combined with small Q waves in leads II and aVF indicates inferior MI. The R wave is somewhat tall in V₂ raising the possibility of posterior wall involvement.

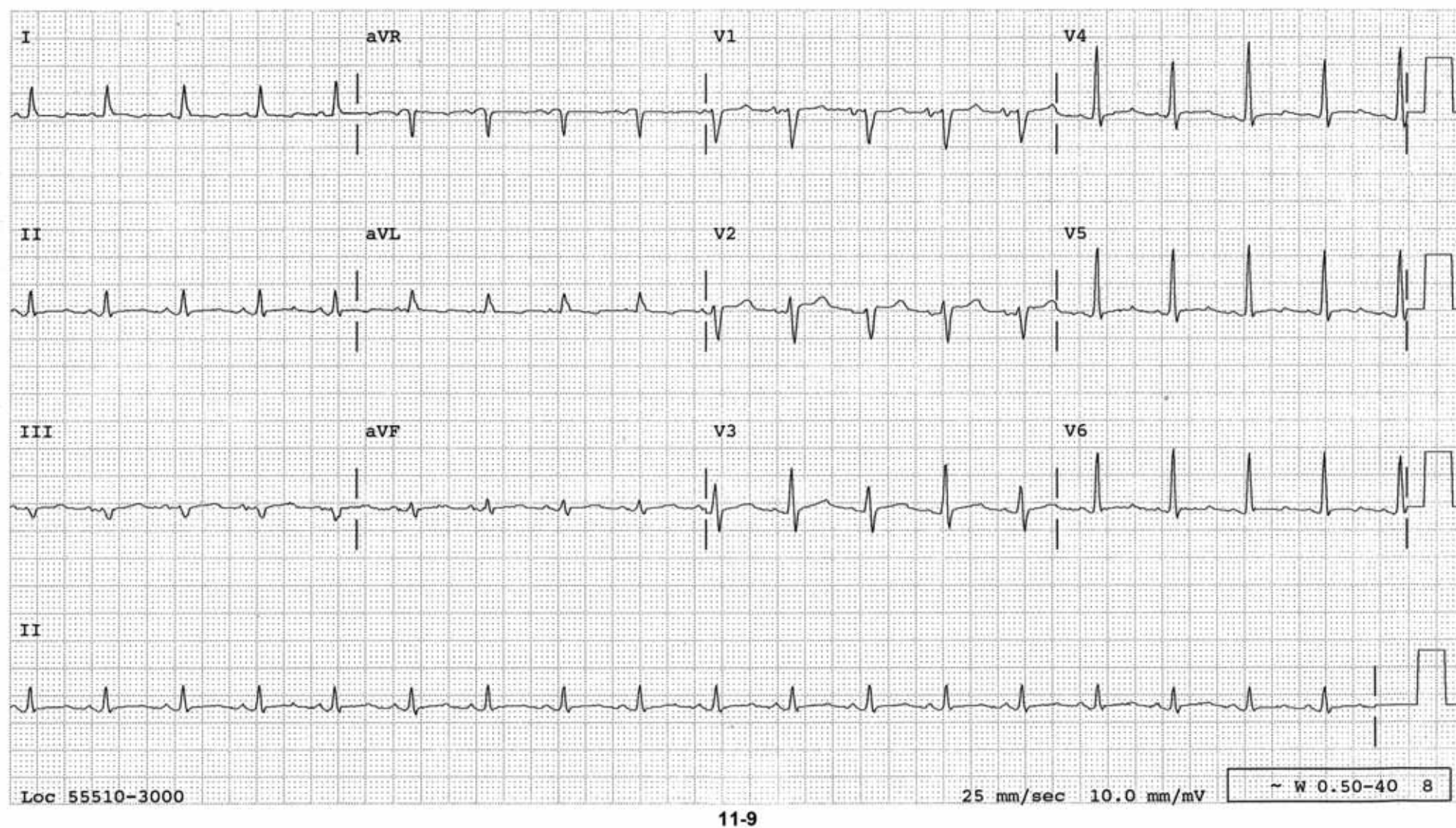
- Dx:*
1. NSR
 2. Inferior infarct, age undetermined
 3. Possible posterior wall involvement



11-8

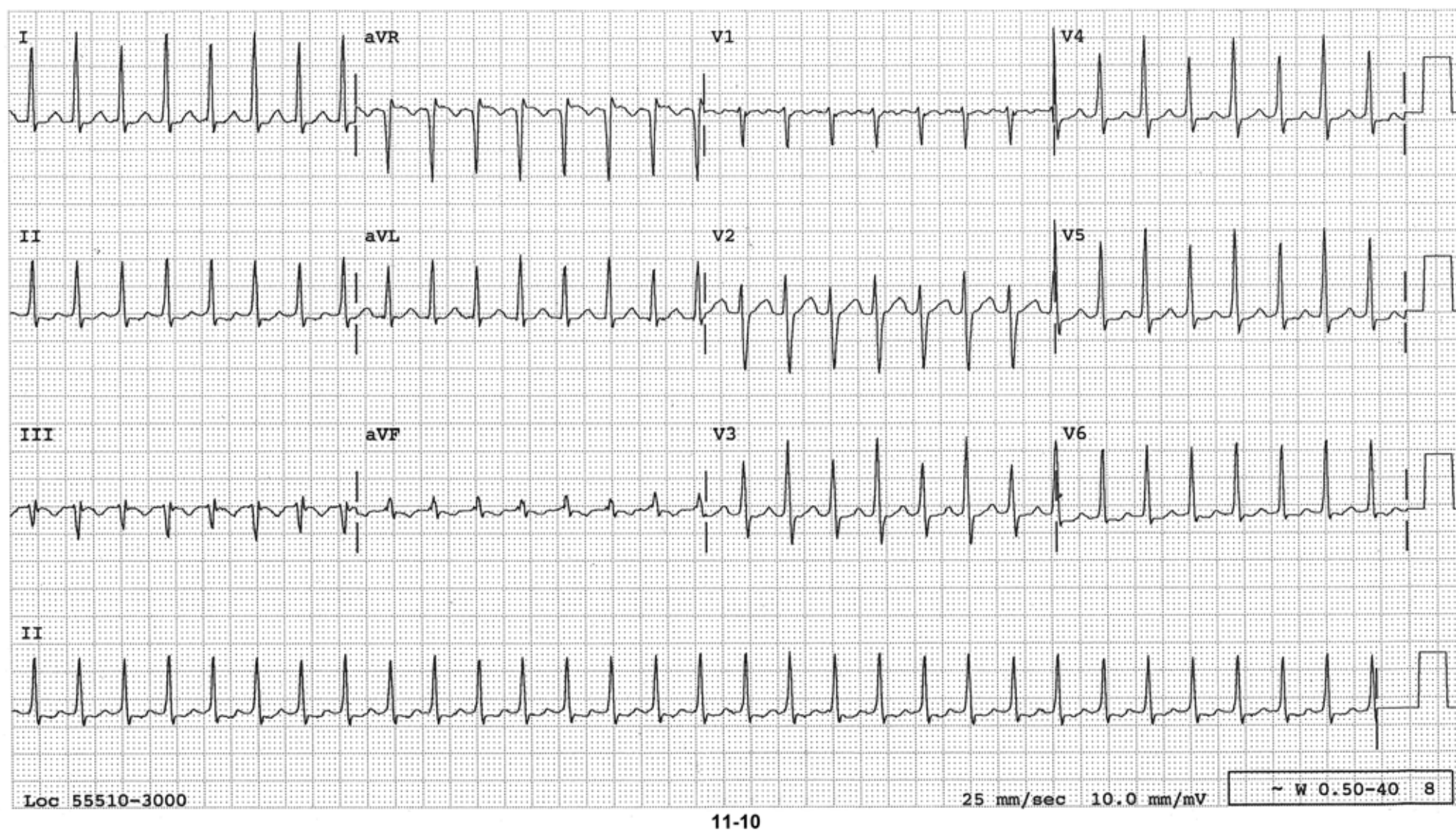
11-8 This is a 12 lead ECG tracing. NSR at a rate of 95/minute. The R waves are tall in the right precordial leads and they regress rather than progress in the left precordial leads. What is happening can be settled by the P wave morphology. Among the six precordial leads, the one with the most biphasic P wave belongs to V_1 , which is V_6 in this case. So, V_4 - V_5 - V_6 is actually V_3 - V_2 - V_1 . How about V_1 - V_3 ? Some might think R waves become taller and taller from V_4 through V_6 . But more often R wave becomes shorter in V_6 than in V_5 . But more convincingly, S wave clearly becomes less and less deep as it progresses from V_4 - V_6 . Based on that, this tracing's V_1 - V_2 - V_3 is actually V_4 - V_5 - V_6 . This is another example where a trivial finding such as P wave morphology can play a decisive role. Increased QRS voltage reflects LVH (the precordial leads are half-standard).

- Dx:
1. NSR
 2. Misplaced precordial leads
 3. LVH



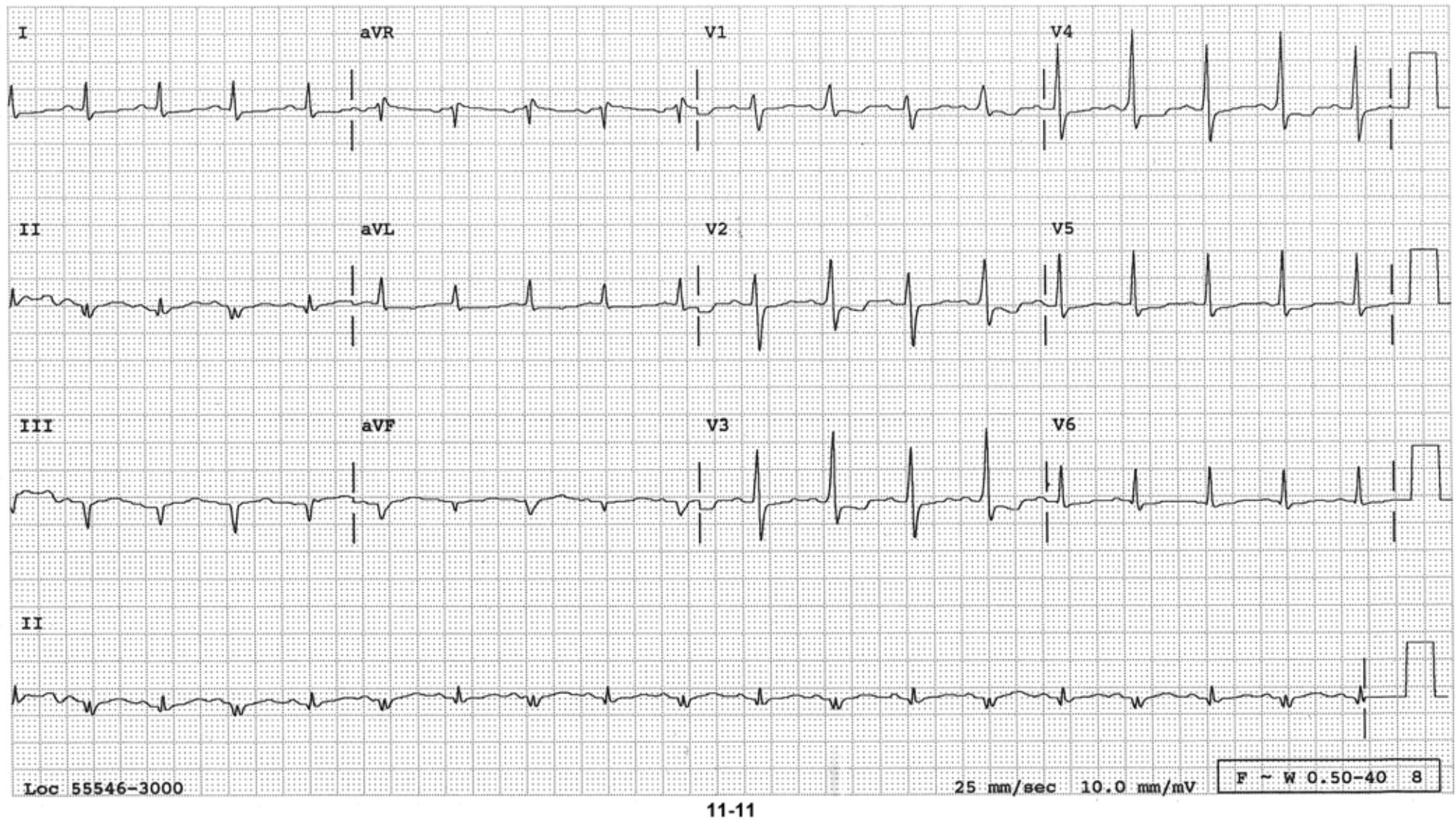
11-9 Sinus tachycardia at a rate of 108/minute. The QRS configuration is alternating in V_3 and V_4 . This is a good example of an electrical alternans (EA). EA during sinus rhythm implies cardiac tamponade.

- Dx:*
1. Sinus tachycardia
 2. Electrical alternans, suggesting cardiac tamponade



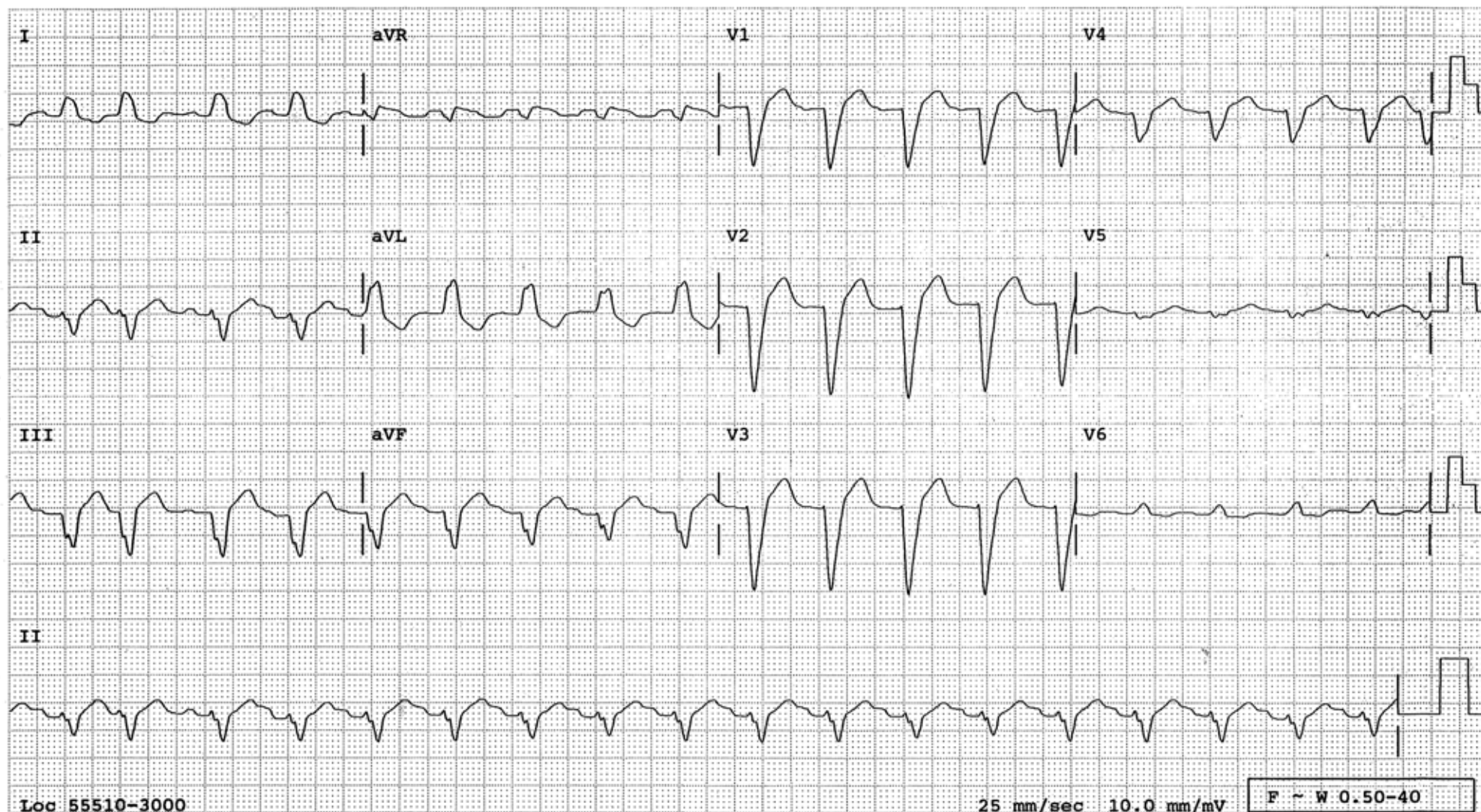
11-10 Narrow-QRS tachycardia at a rate of 185/minute and is a good example of SVT. Electrical alternans (EA) is present in many leads. EA is well-known to be present in some cases of SVT, atrial flutter, or VT without any pericardial problems. EA only during sinus rhythm implies cardiac tamponade.

- Dx:*
1. SVT
 2. Electrical alternans



11-11 Sinus tachycardia at a rate of 109/minute. QRS complexes alternate in many leads suggesting electrical alternans (EA). This is an example of preexcitation affecting only every other beat, simulating EA. A short P-R interval with a delta wave in every other QRS complex is better appreciated in V₄. Preexcitation of every other beat may also simulate ventricular bigeminy if the affected QRS complex is noticeably wide.

- Dx:*
1. Sinus tachycardia
 2. Preexcitation of every other beat simulating EA

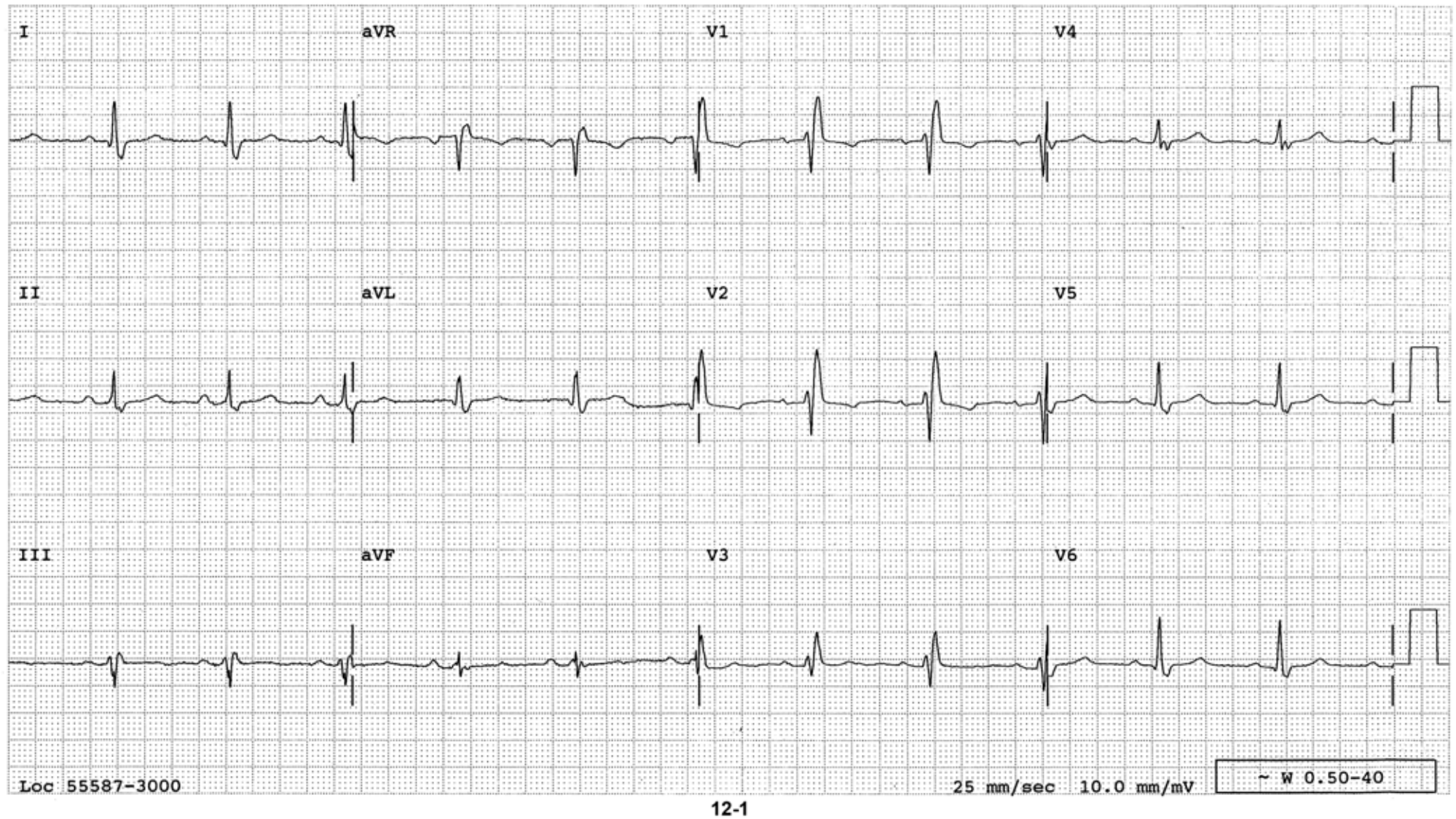


11-12

11-12 A wide QRS tachycardia at a rate of 115/minute. QRSs are wide and have a typical LBBB pattern. In the precordial leads, the P waves are not readily recognizable. In the rhythm strip of lead II, one could suspect a P wave after the T wave. This is confirmed by the compensatory pause after a premature complex (the second complex) which allows us to see the P wave convincingly. Therefore, this is an example of sinus tachycardia in a patient who has LBBB, and not VT or junctional rhythm. Another example of the usefulness of a premature beat.

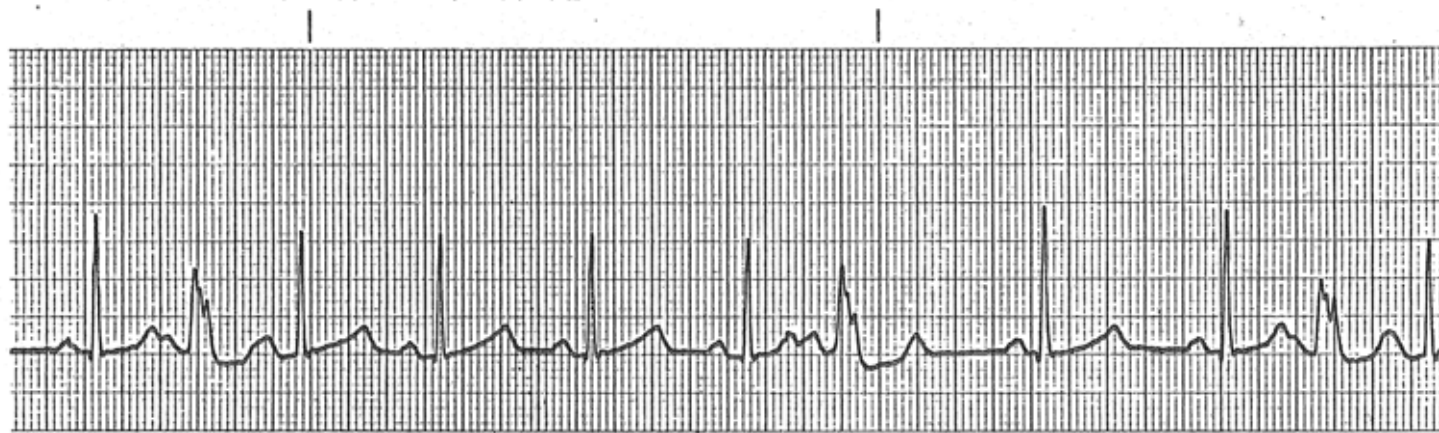
- Dx:
1. Sinus tachycardia
 2. LBBB
 3. Supraventricular premature complex

SECTION 12



12-1 Normal sinus rhythm at a rate of 70/minute. A typical RBBB pattern (rsR' in V_1 and broad S waves in leads I, aVL and V_6) is present.

- Dx: 1. NSR
2. RBBB

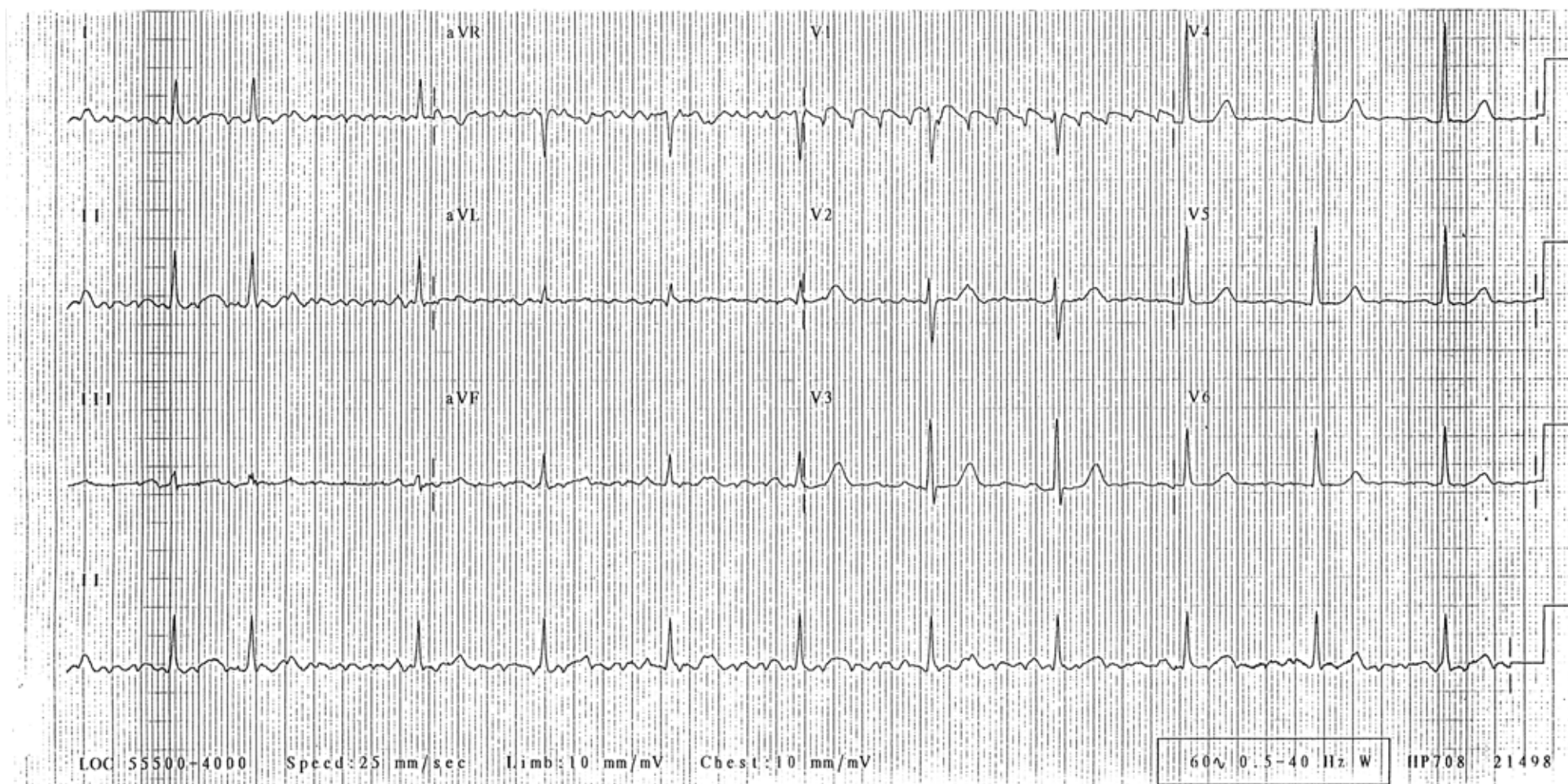


12-2

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- 12-2 Normal sinus rhythm at a rate of 65/minute. Three wide QRS complexes are present. They are preceded by a P wave which occurs prematurely indicating that these are atrial premature complexes with aberrant conduction rather than ventricular premature complexes.

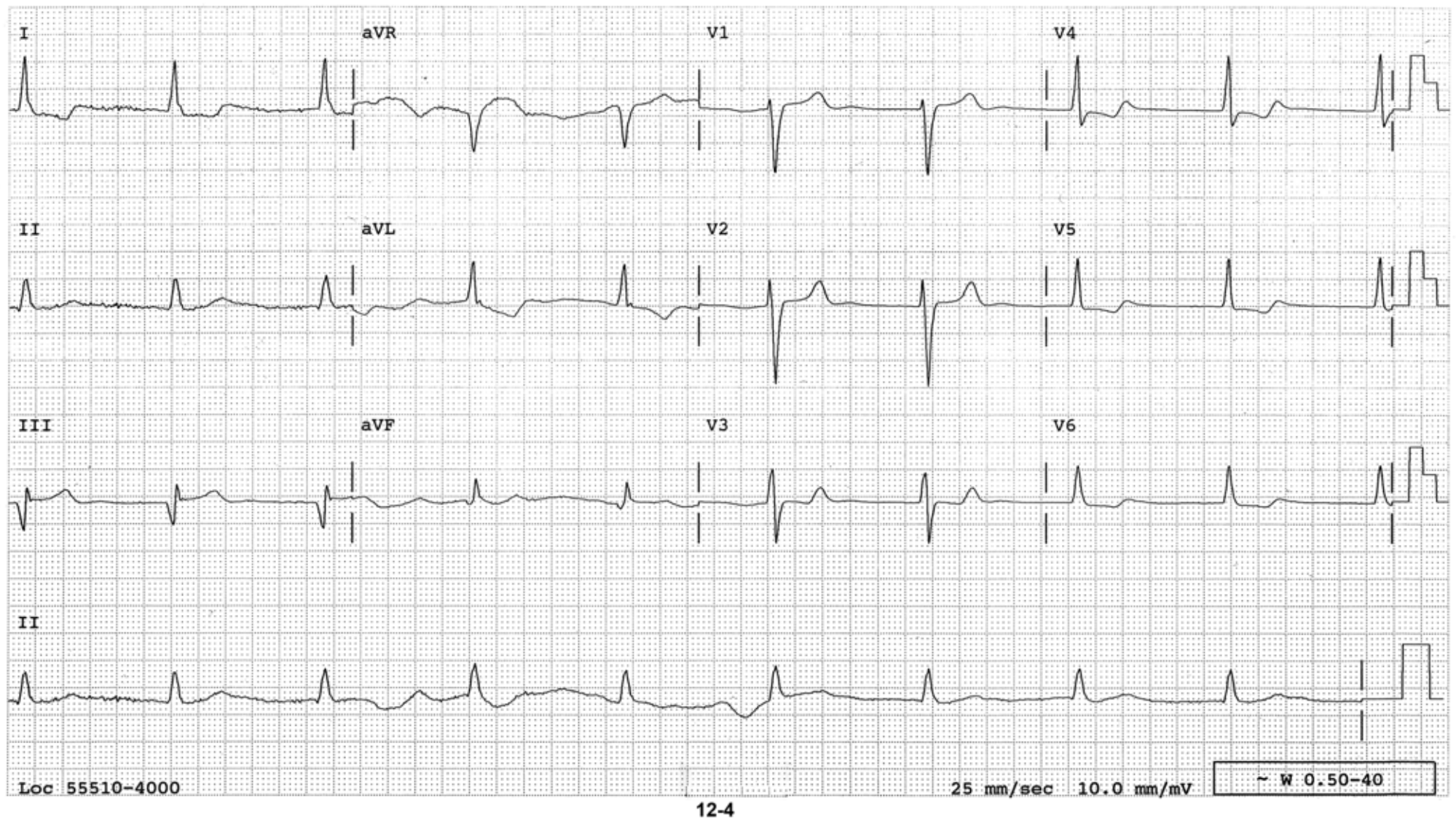
Dx: NSR with a frequent PACs with aberrant conduction



12-3

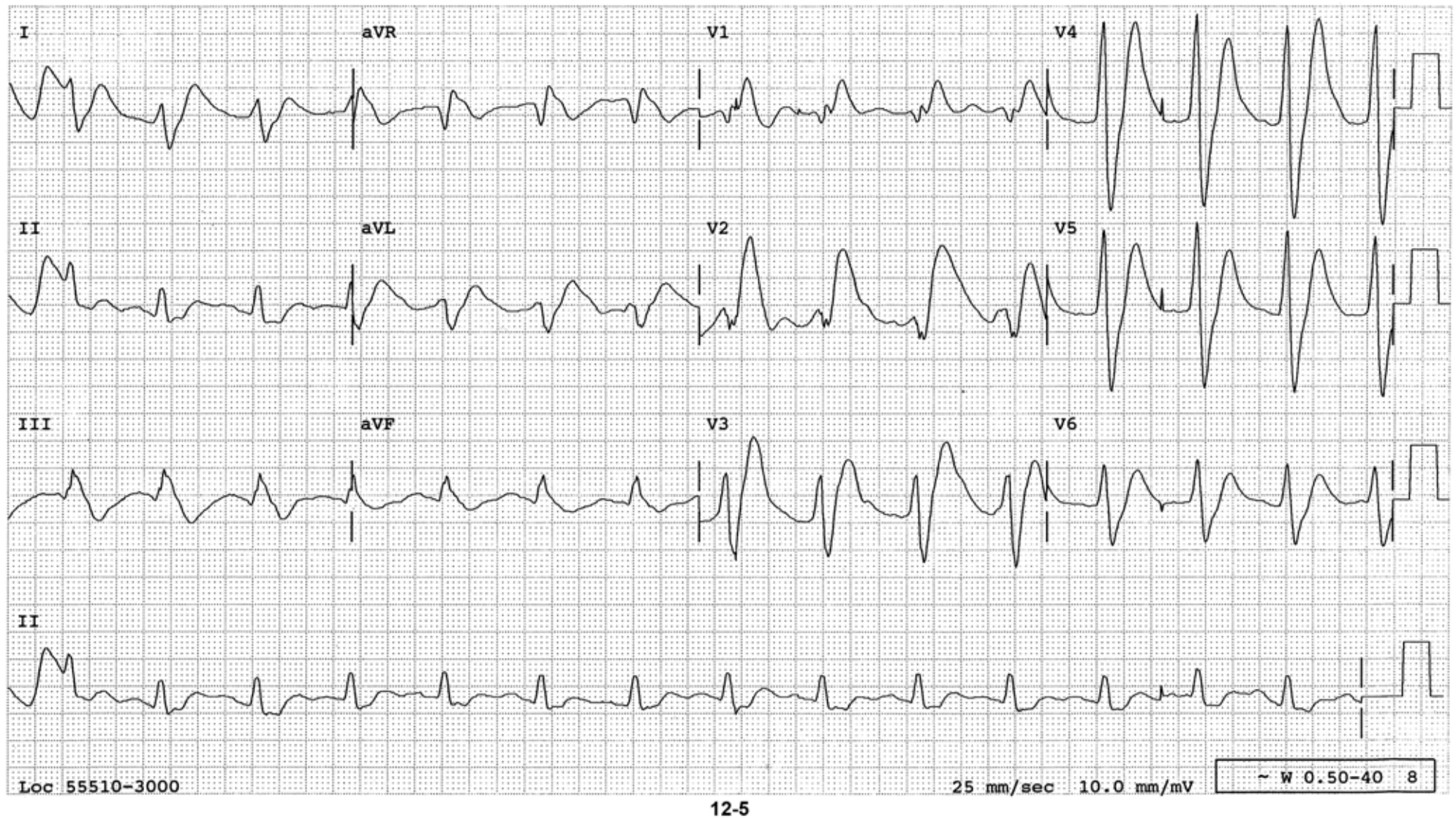
12-3 There is a regular rhythm with narrow QRSs at a rate of 65/minute. The findings in V_1 certainly suggest atrial flutter waves. However, other leads such as III or V_3 convincingly reveal normal P waves indicating that the patient is in NSR. If this were atrial flutter, regularly occurring QRS complexes mean a fixed AV conduction ratio such as 4:1 or 5:1, etc. In that case, the flutter waves and the QRS complexes should have maintained a fixed relationship, which is not the case here. So there are many clues that this is not atrial flutter. In fact, this tracing was taken from a patient who was shivering.

- Dx:
1. NSR
 2. One atrial premature complex
 3. Muscle tremor simulating atrial flutter



12-4 A regular rhythm at a rate of 54/minute. No P waves can be definitely identified. The QRSs are wide but not wide enough to make one think of ventricular rhythm and this is an accelerated AV junctional rhythm. Note that the precordial leads are half-standard and the voltage criteria and ST-T changes for LVH are present. In fact, the QRS widening may be secondary to LVH. The Q wave is prominent in lead III with some ST elevation indicating inferior MI, which may be the cause of accelerated junctional rhythm.

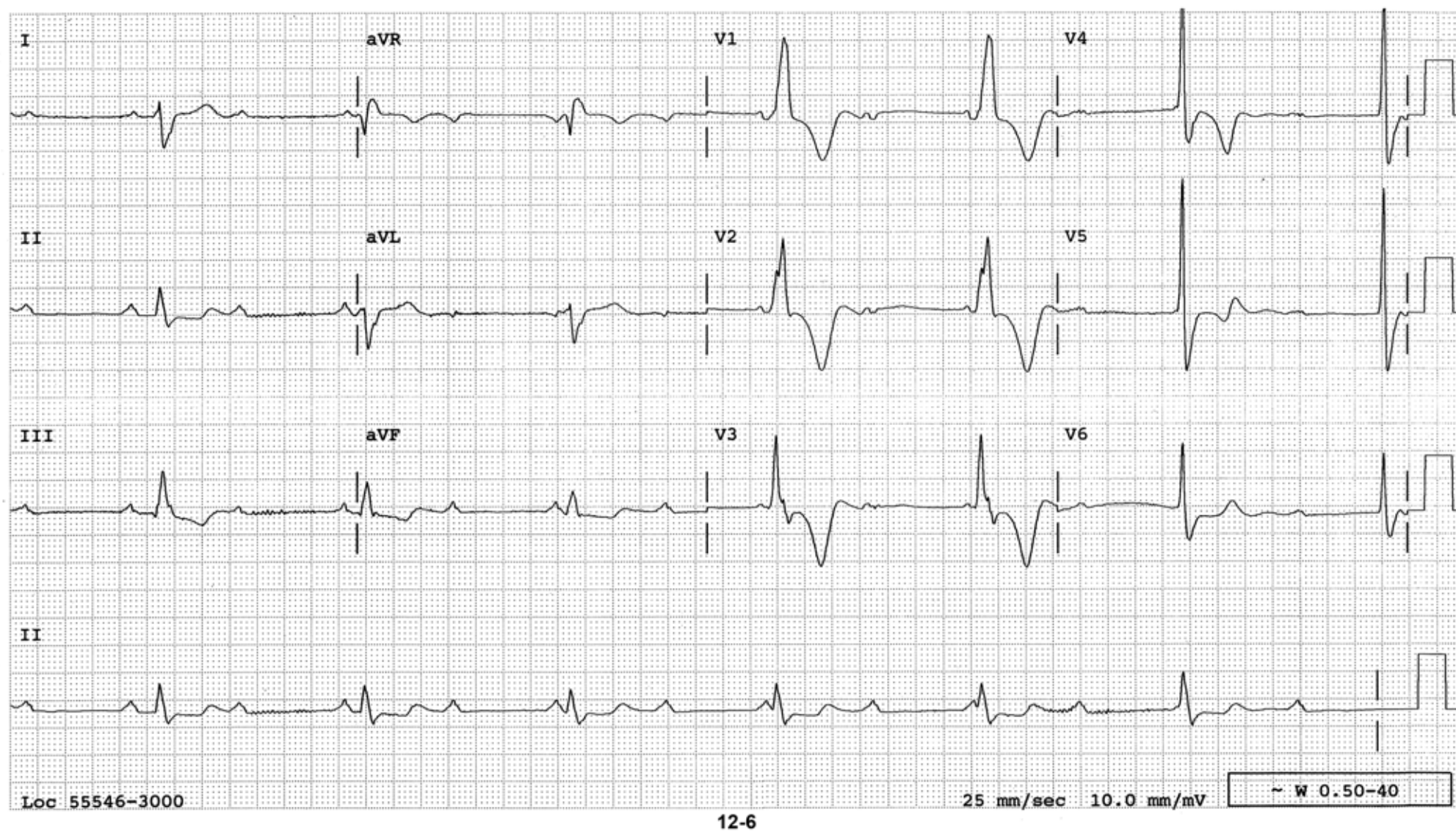
- Dx:
1. Accelerated AV junctional rhythm
 2. Inferior infarct, probably recent
 3. LVH



12-5 A regular rhythm at a rate of 87/minute. P waves are not easily recognizable except in the rhythm strip of lead II. Even then, the P wave amplitude is diminished. QRSs are wide and have RBBB features. The T waves are tall, pointed and peaked especially in V_4 . These are features of hyperkalemia. In V_1 - V_2 , the ST-segment begins from the R' and is downsloping. These are acquired Brugada ECG pattern from hyperkalemia, which transiently disables the Na channel causing this ECG changes.

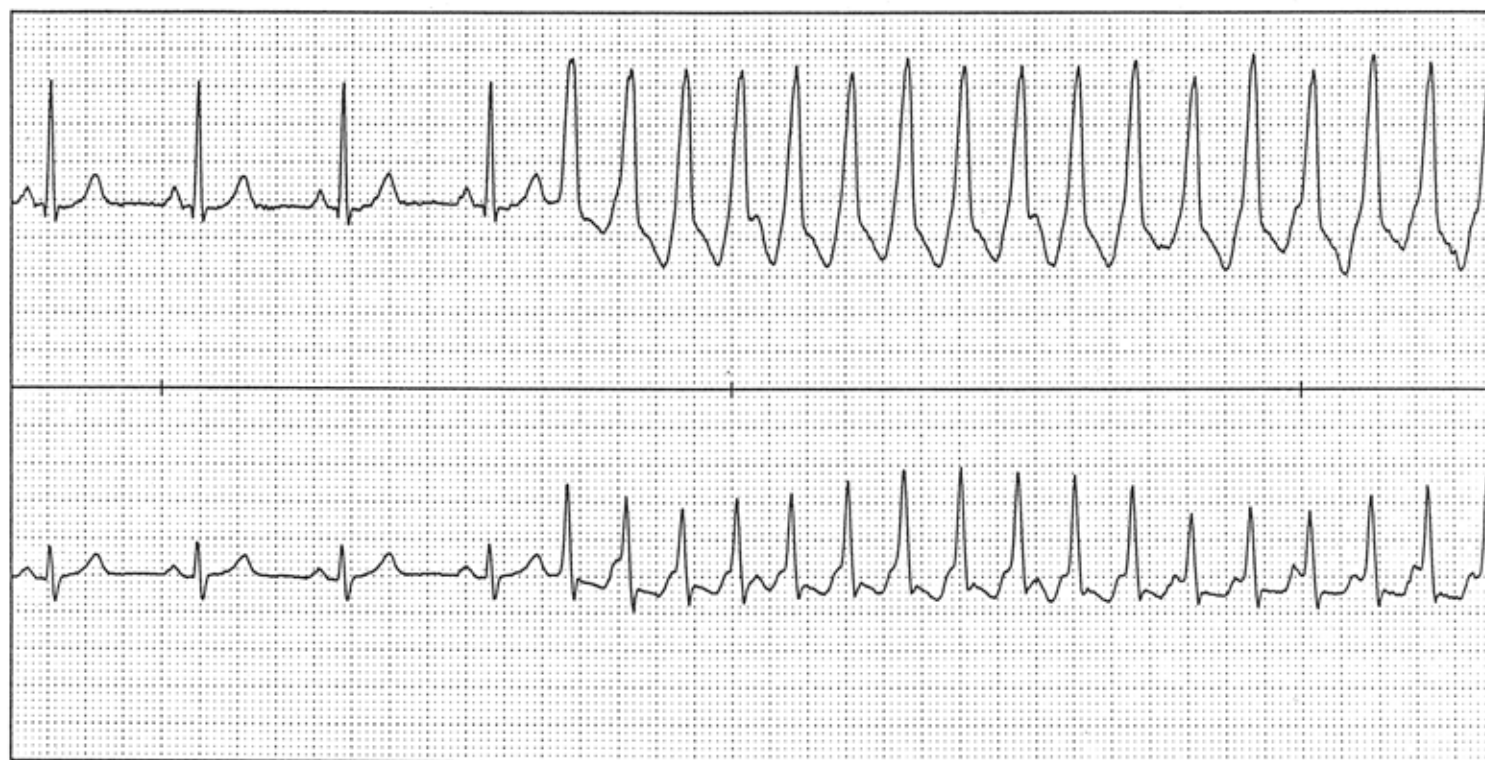
Dx: 1. NSR

2. Findings highly suggestive of hyperkalemia with acquired Brugada ECG pattern



12-6 QRSs occur regularly at a rate of 40/minute. There are more P waves than QRSs and the regularly occurring P and QRS have no fixed relationship indicating complete AV block. The QRS is wide and has bifascicular block pattern (RBBB + left posterior fascicular block). The escape mechanism may be originating from the ventricle or from the AV junction in a patient with preexisting BIFB. This question cannot be settled from the tracing provided.

Dx: NSR and 3° AV block with either ventricular or junctional escape rhythm in a patient with BIFB



12-7

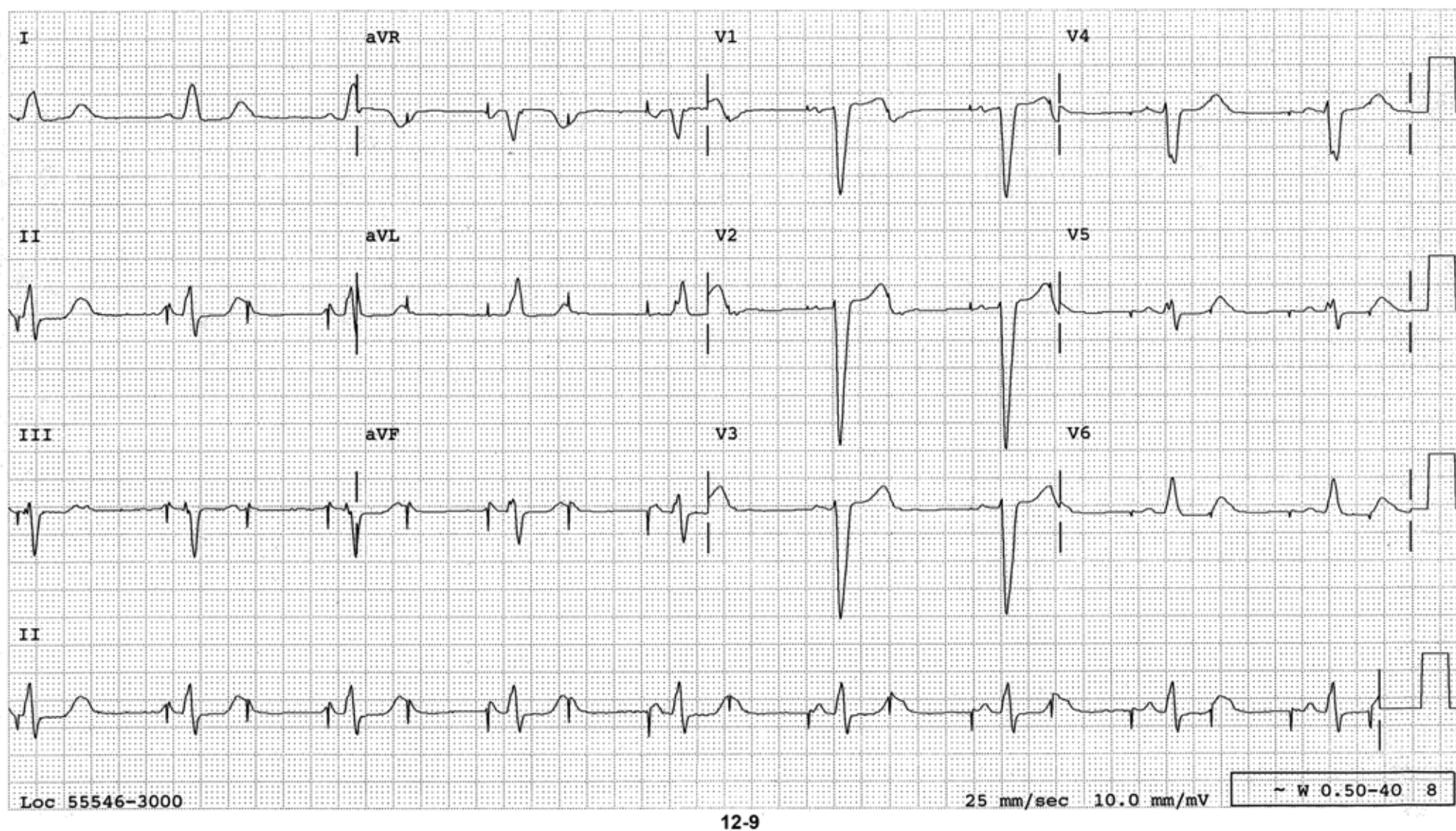
- 12-7 Regular sinus rhythm is present initially which is followed by a wide QRS tachycardia at a rate of about 180/minute. In a patient who is in sinus rhythm, when the beginning of the wide QRS tachycardia is included in the tracing, there should be little difficulty deciding whether this is a run of VT or SVT with aberrant conduction. The first complex of this wide QRS tachycardia is not preceded by a P wave, proving that it is a ventricular premature complex; hence, this is a run of VT.

Dx: NSR followed by VT



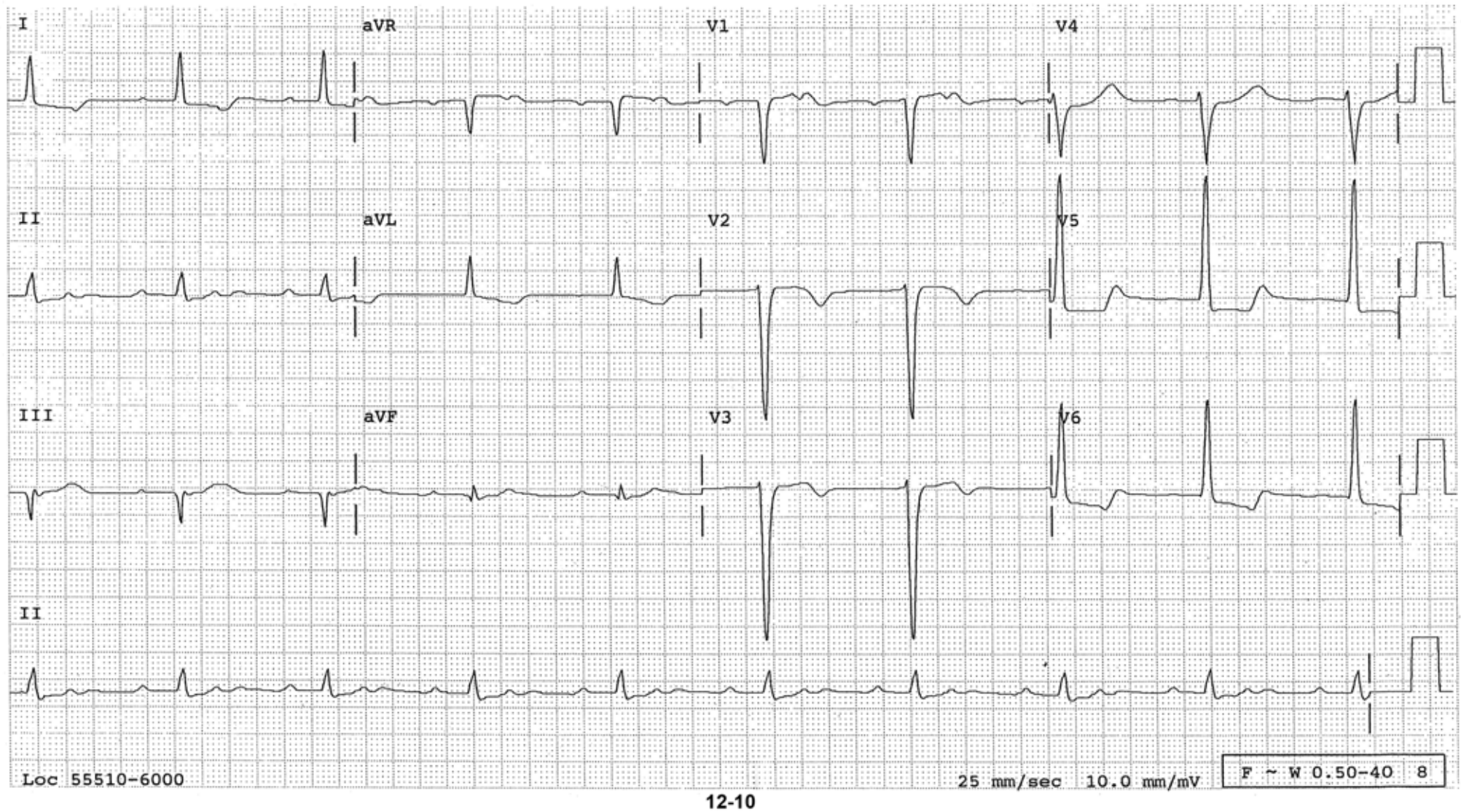
12-8 A regular rhythm with narrow QRSs at a rate of 120/minute. No P waves can be identified. The rhythm is accelerated junctional rhythm. The QRS voltage is low in the limb leads. There is a Q wave in lead III with a slight ST elevation indicating inferior MI. The R waves are tall in the right precordial leads. These, combined with horizontal ST depression, reflect reciprocal changes of posterior MI.

- Dx:
1. Accelerated junctional rhythm
 2. Acute inferoposterior infarct
 3. Low QRS voltage



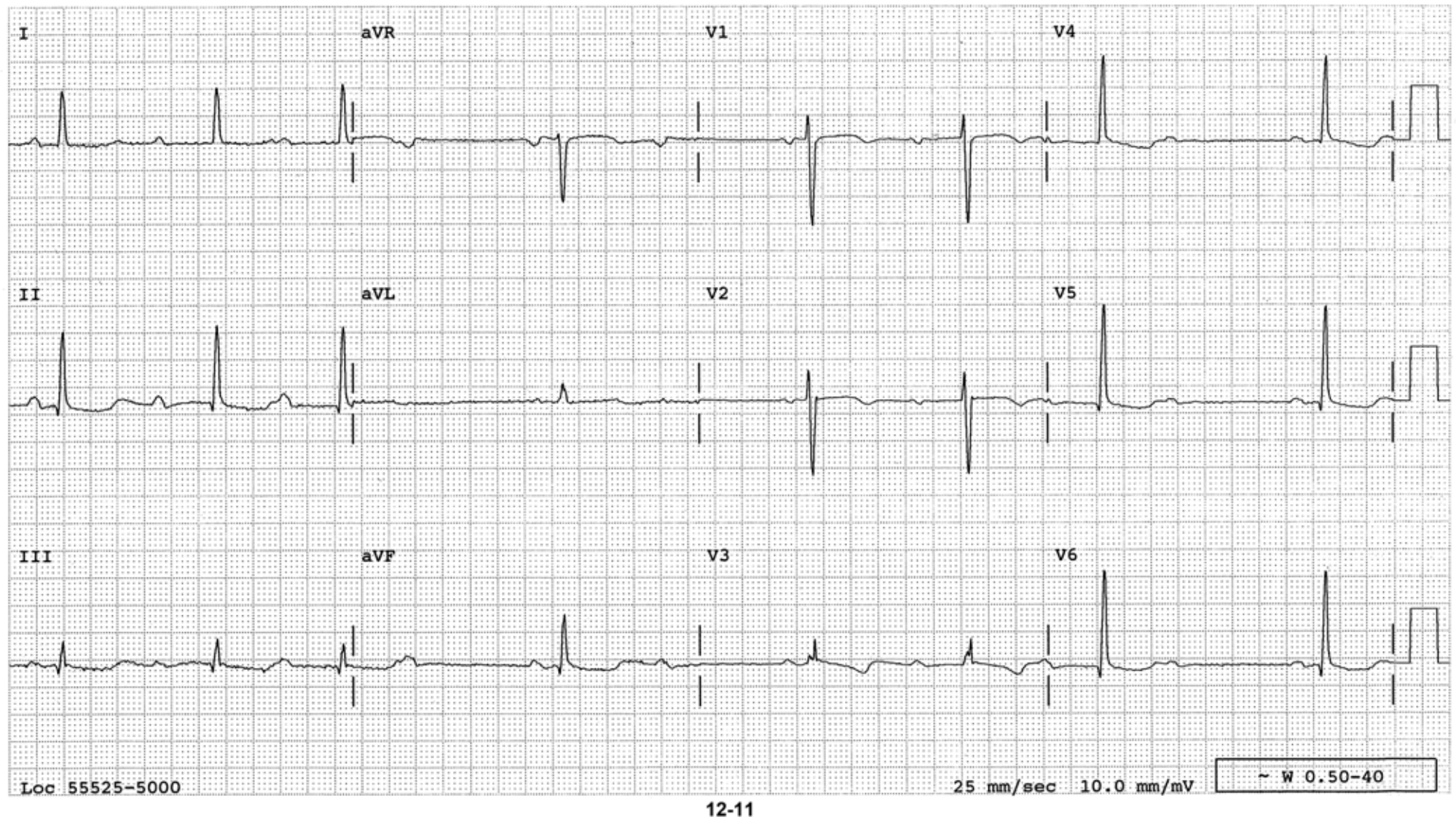
12-9 Pacemaker spikes occur regularly (see the rhythm strip of lead II). Careful examination reveals that the pacemaker spikes do not result in either the P wave or the QRS indicating pacemaker failure to capture. This will result when the pacing electrode is not in optimal contact with the endocardium. The patient is in sinus rhythm. Leads V_1 - V_3 and III suggest that there is another P wave between the QRSs with 2:1 AV conduction. The QRSs are wide and have typical LBBB pattern.

- Dx:
1. NSR with 2:1 AV block
 2. LBBB
 3. Pacemaker failure to capture



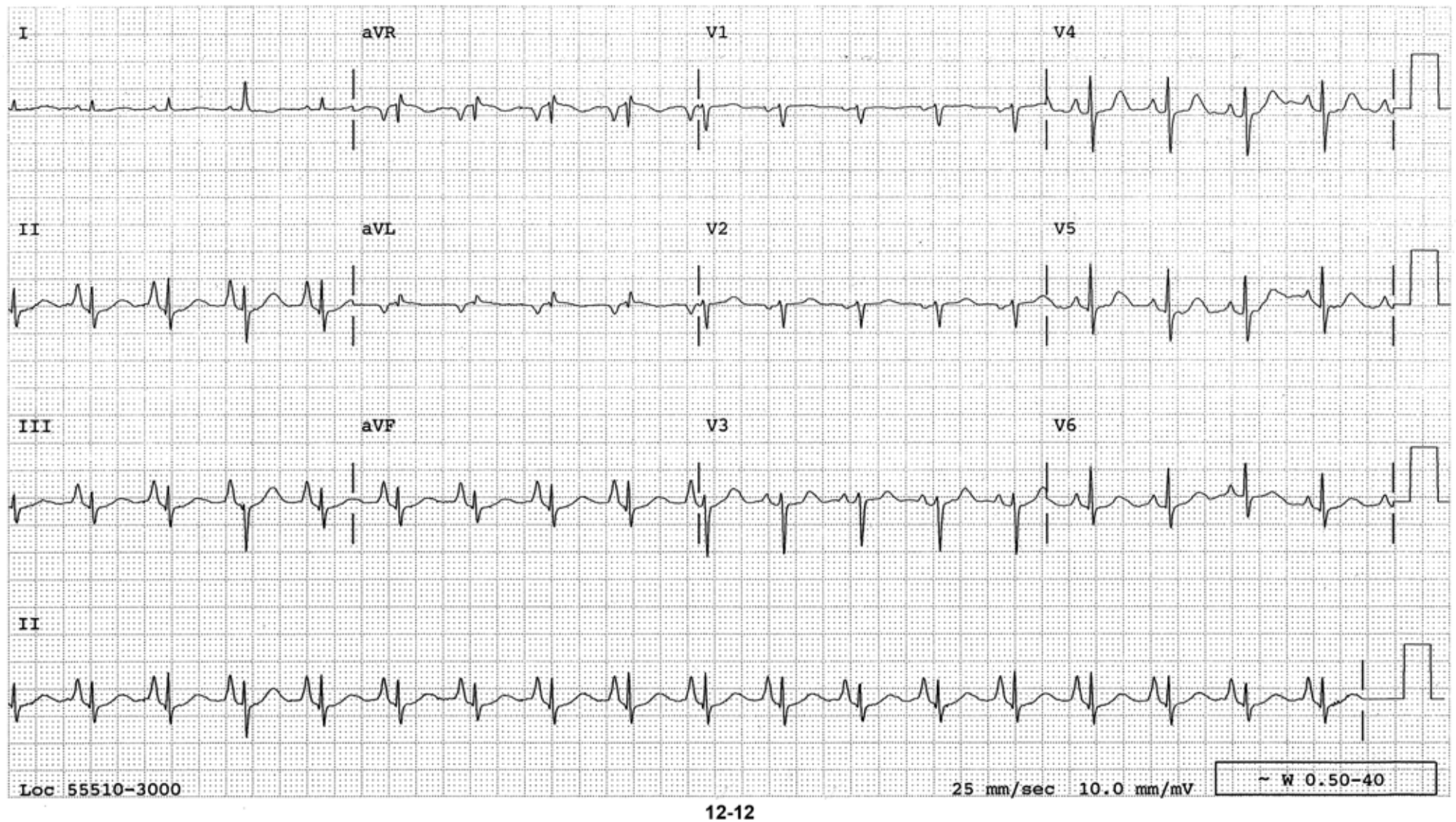
12-10 A regular rhythm at a rate of 60/minute. P waves are recognizable in front of each QRS with a long P-R interval. Careful examination of the lead II rhythm strip reveals another P wave between the QRSs. These P waves march out indicating sinus tachycardia at a rate of 115/minute with 2:1 AV block. Voltage criteria and ST-T changes for LVH are present. A prominent Q wave with a small R wave in lead III indicates old inferior MI.

- Dx:
1. Sinus tachycardia with 2:1 AV block
 2. LVH
 3. Old inferior infarct



12-11 The QRSs are occurring not regularly. The P-R interval progressively lengthens and finally the fourth P wave is blocked. The fifth P wave conducts with a shorter P-R interval and the following P wave again is blocked. The findings are typical of Type I 2° AV block. The momentary 2:1 AV block in the middle of the strip is not Type II 2° AV block as long as there are findings of Wenckebach phenomenon at other parts of the tracing. The Q-T interval is markedly prolonged.

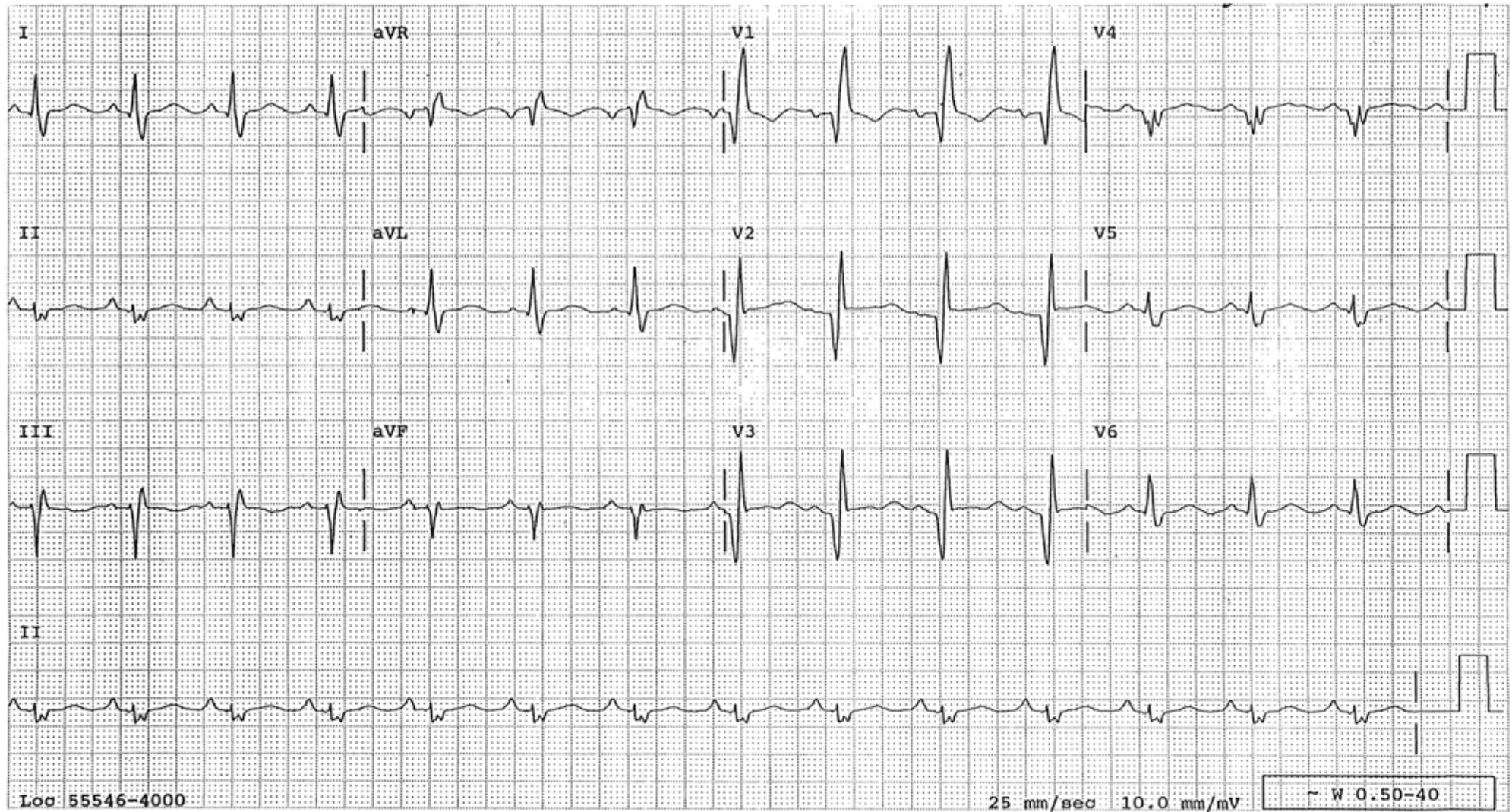
- Dx:*
1. Sinus rhythm
 2. Type I 2° AV block
 3. Long Q-T interval



12-12 Sinus tachycardia at a rate of 106/minute. The P waves are very tall measuring about 5 mm in lead II indicating RAE. There is a poor progression of the R waves in the right precordial leads and S waves in the left precordial leads raising the possibility of chronic obstructive lung disease pattern.

- Dx:*
1. Sinus tachycardia
 2. RAE
 3. Consider chronic obstructive lung disease

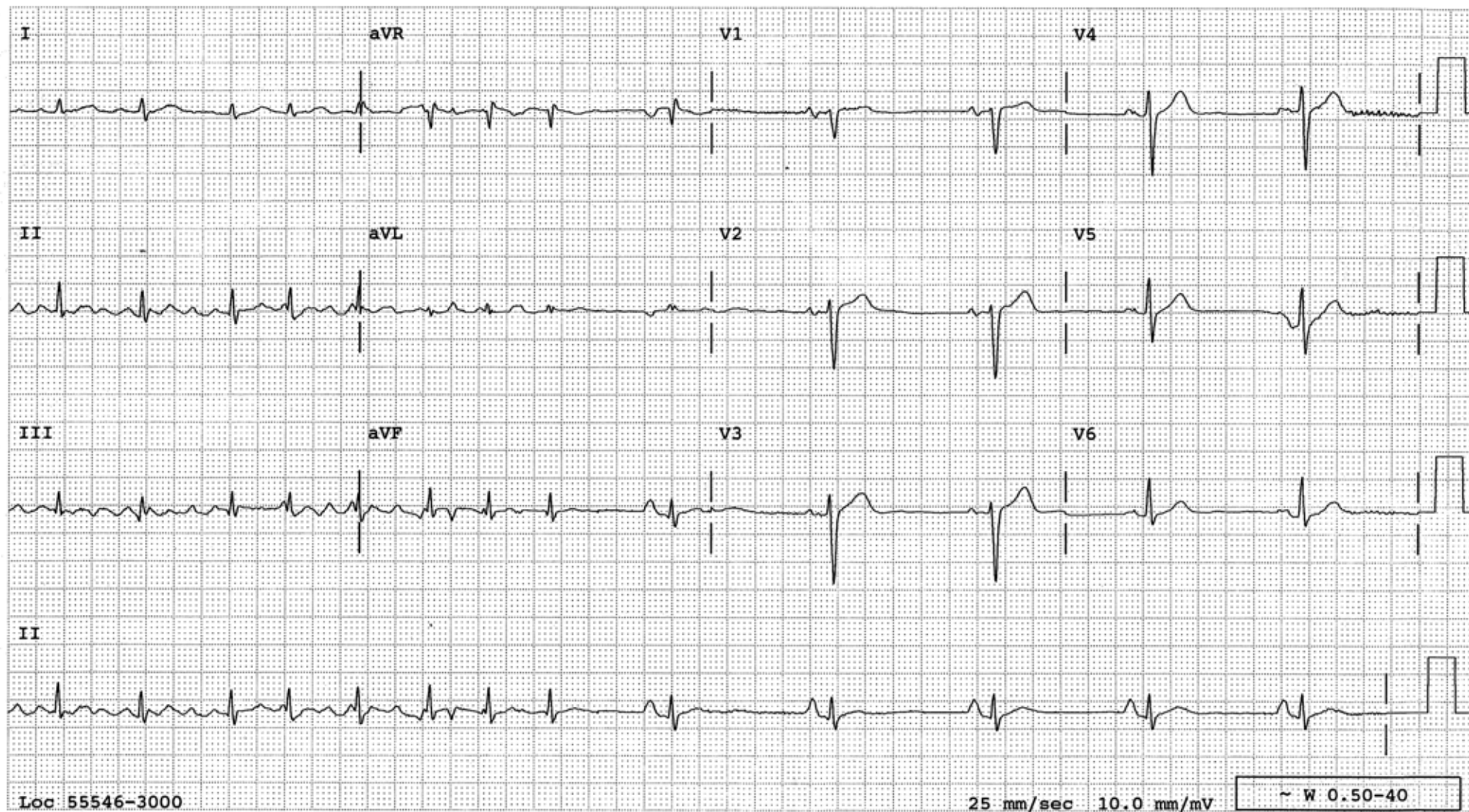
SECTION 13



13-1

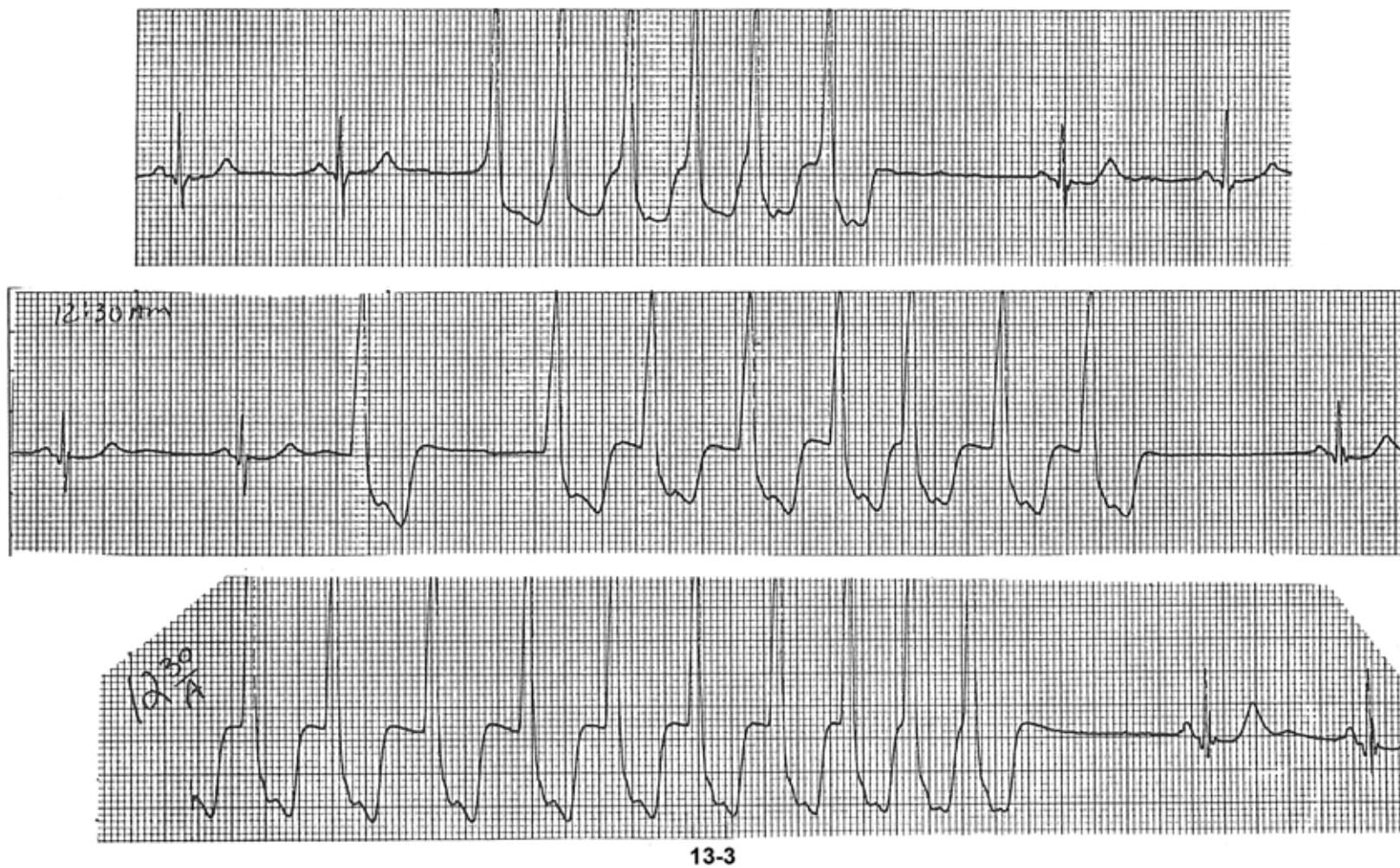
13-1 Normal sinus rhythm at a rate of 80/minute. RBBB is present. In the right precordial leads, the initial R wave (the first “rabbit ear”) of the rsR’ is missing due to anteroseptal MI. RBBB affects only the terminal portion of the QRS and it does not interfere with the manifestations of a Q wave infarction.

- Dx:*
1. NSR
 2. RBBB
 3. Anteroseptal infarct, probably old



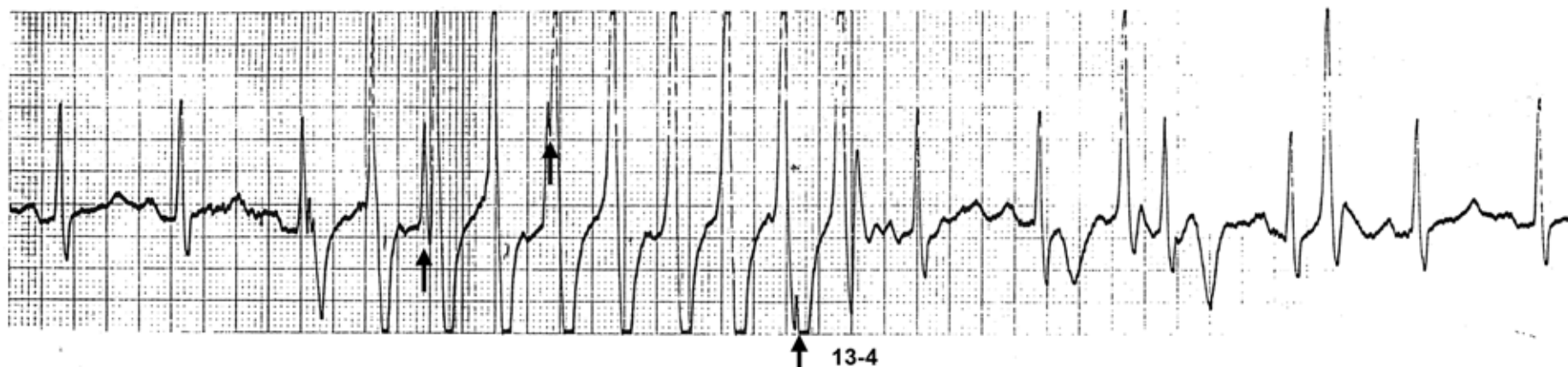
13-2 Atrial fibrillation is present in the first part of the tracing. The rhythm spontaneously converts to NSR in the latter part of the tracing. The QRS voltage is low in the limb leads. The P wave is tall in lead II indicating RAE.

- Dx:
1. Atrial fibrillation with spontaneous conversion to NSR
 2. Low QRS voltage
 3. RAE



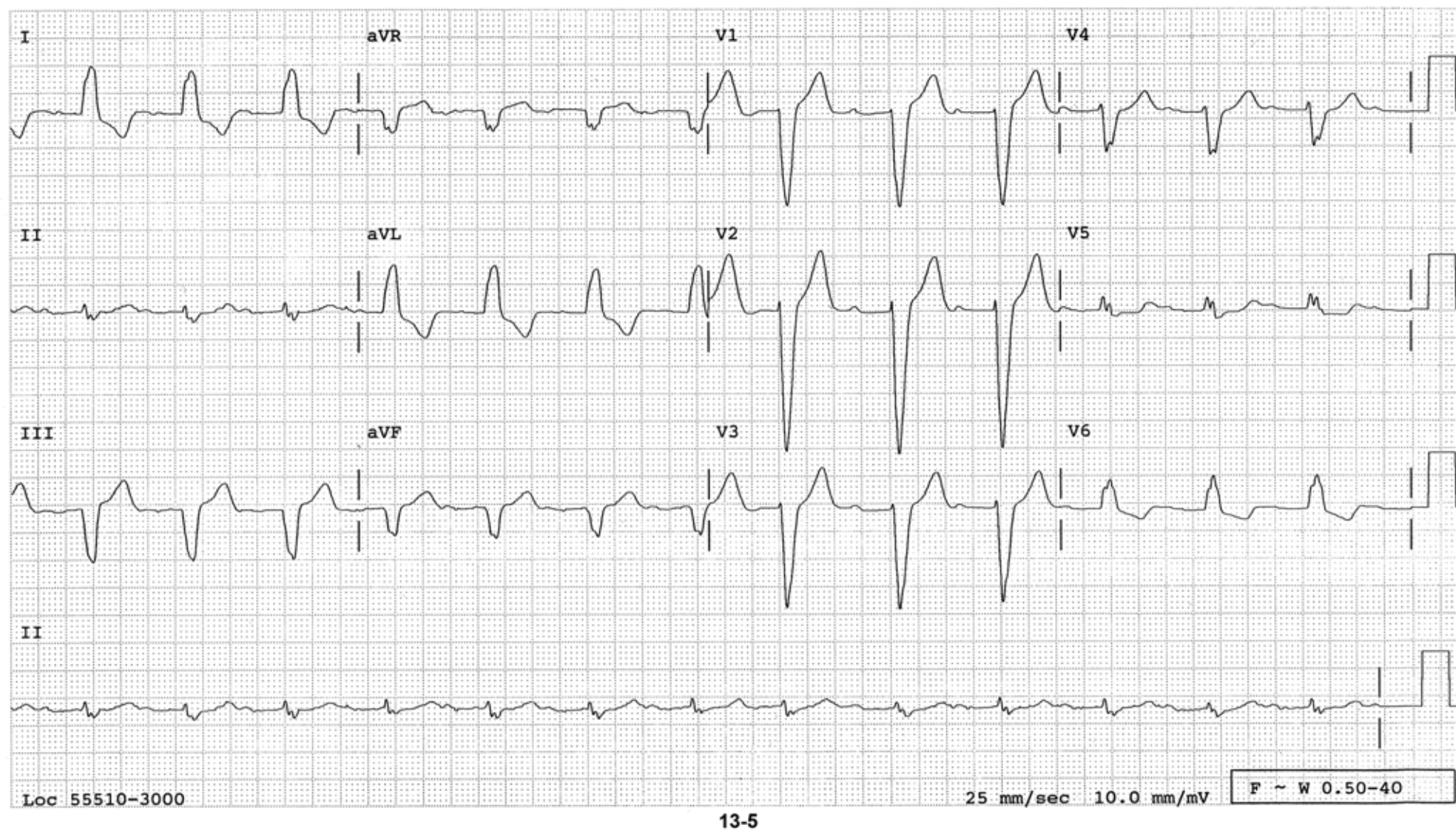
13-3 Rhythm strips reveal NSR with runs of wide QRS tachycardia. The beginning of this wide QRS tachycardia is not preceded by a P wave proving that these are runs of VTs. VT can be this much irregular.

Dx: Sinus rhythm with short runs of VT



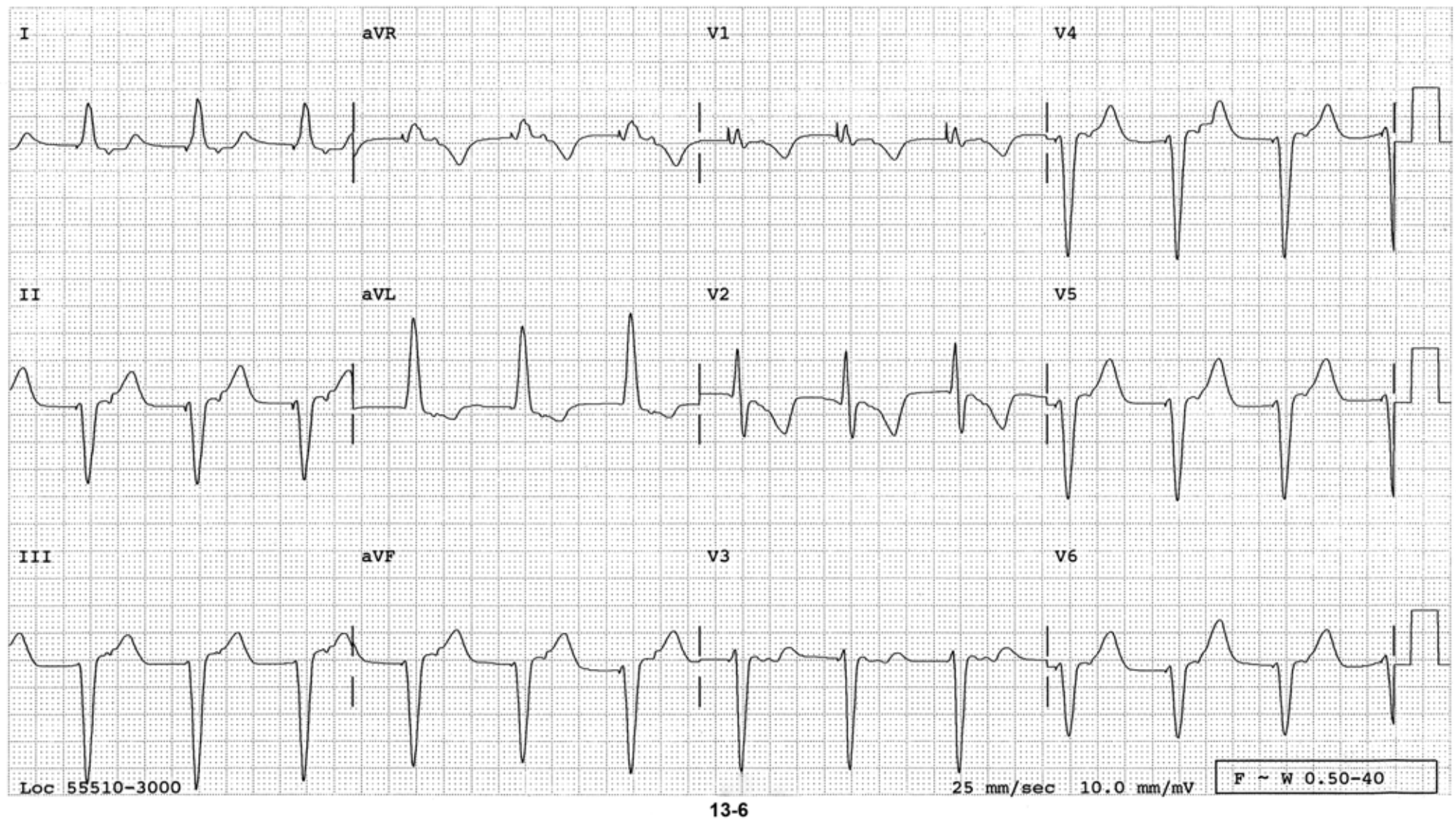
13-4 Normal sinus rhythm is present at the beginning. In the middle of the strip, there appears to be a wide QRS tachycardia at a rate of about 160/minute. A careful examination reveals that the regularly occurring QRSs can be seen at times (↓) indicating that these are artifacts superimposed upon the regular sinus rhythm. The last two wide complexes do not interrupt the regularity of the basic sinus rhythm, again supporting the contention that these are artifacts.

- Dx:*
1. NSR
 2. Artifacts mimicking a run of VT or PVCs



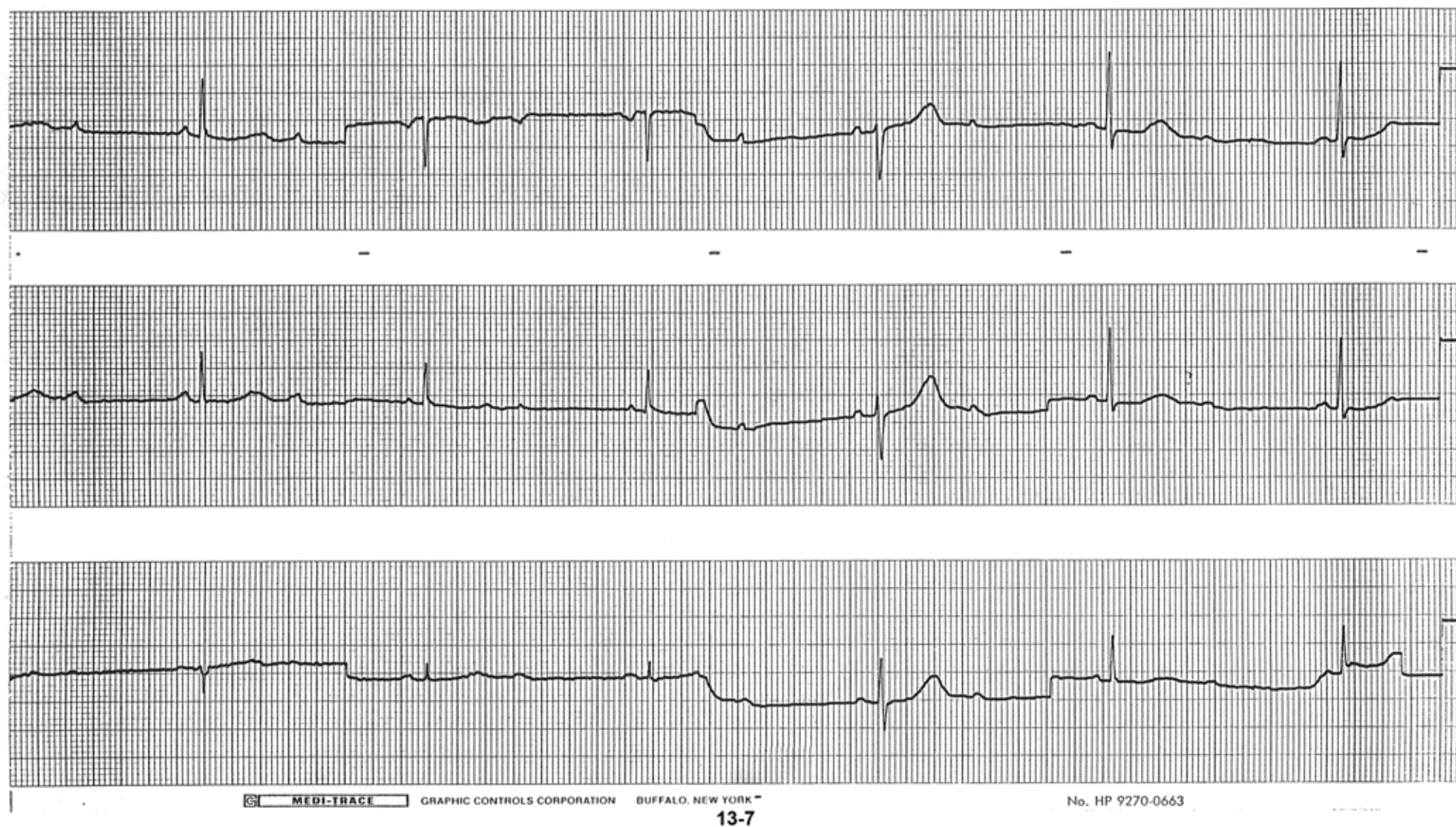
13-5 Normal sinus rhythm at a rate of 81/minute. The P-R interval is prolonged to 242 milliseconds indicating 1° AV block. The QRSs are wide and have typical LBBB pattern.

- Dx:
1. NSR
 2. 1° AV block
 3. LBBB



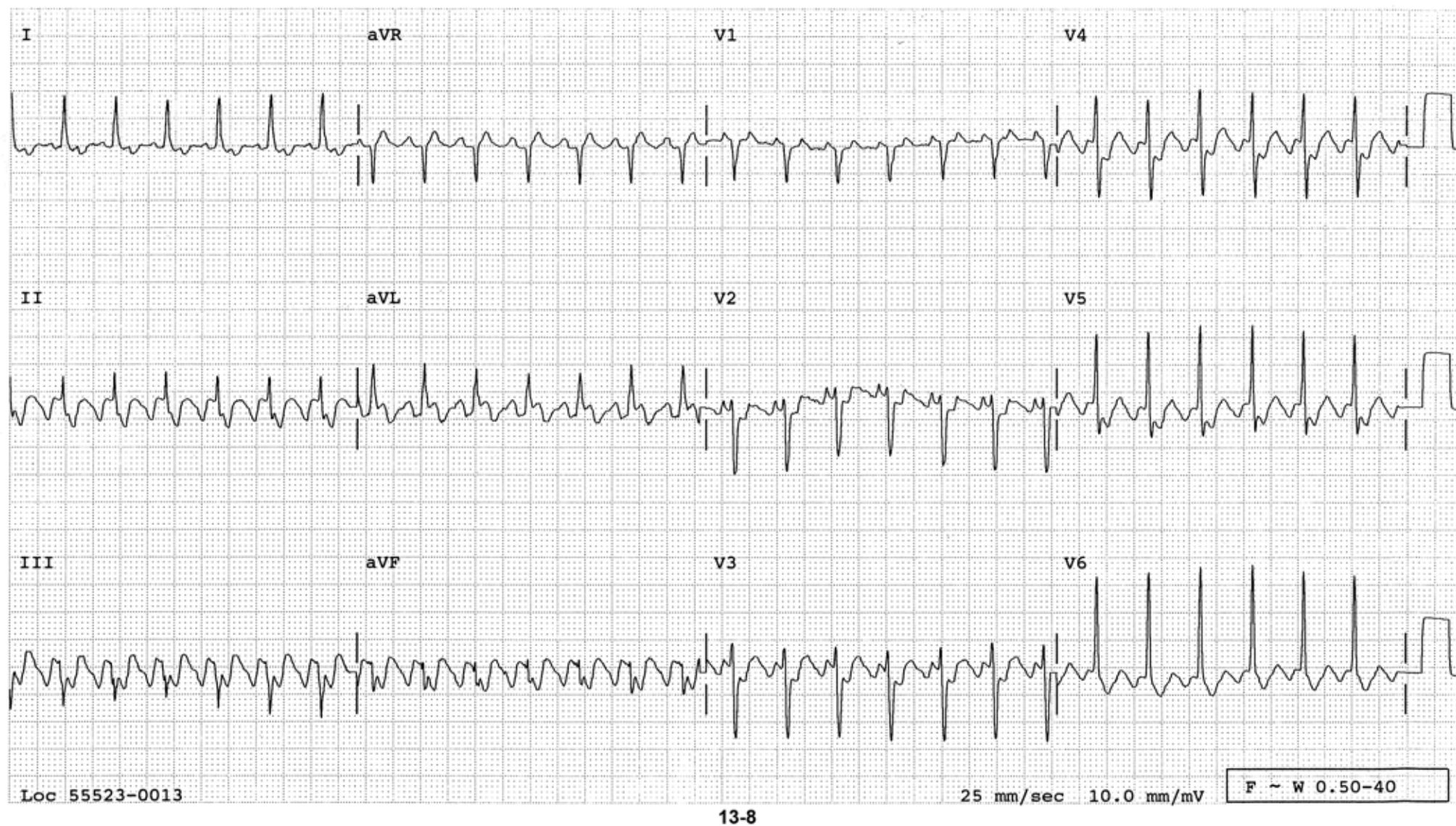
13-6 A regular rhythm at a rate of 75/minute. Pacemaker spikes are easily recognizable in some leads (leads I, II, V₁ and V₄-V₆). The upstroke of the T wave is distorted with a negative wave, which is a retrograde P wave. This 1:1 ventriculoatrial (VA) conduction causing the atria to contract during ventricular systole sometimes (~20%) cause a pacemaker syndrome (pulsating feeling in the neck, dizziness and sometimes syncope).

Dx: Ventricular paced rhythm with 1:1 VA conduction



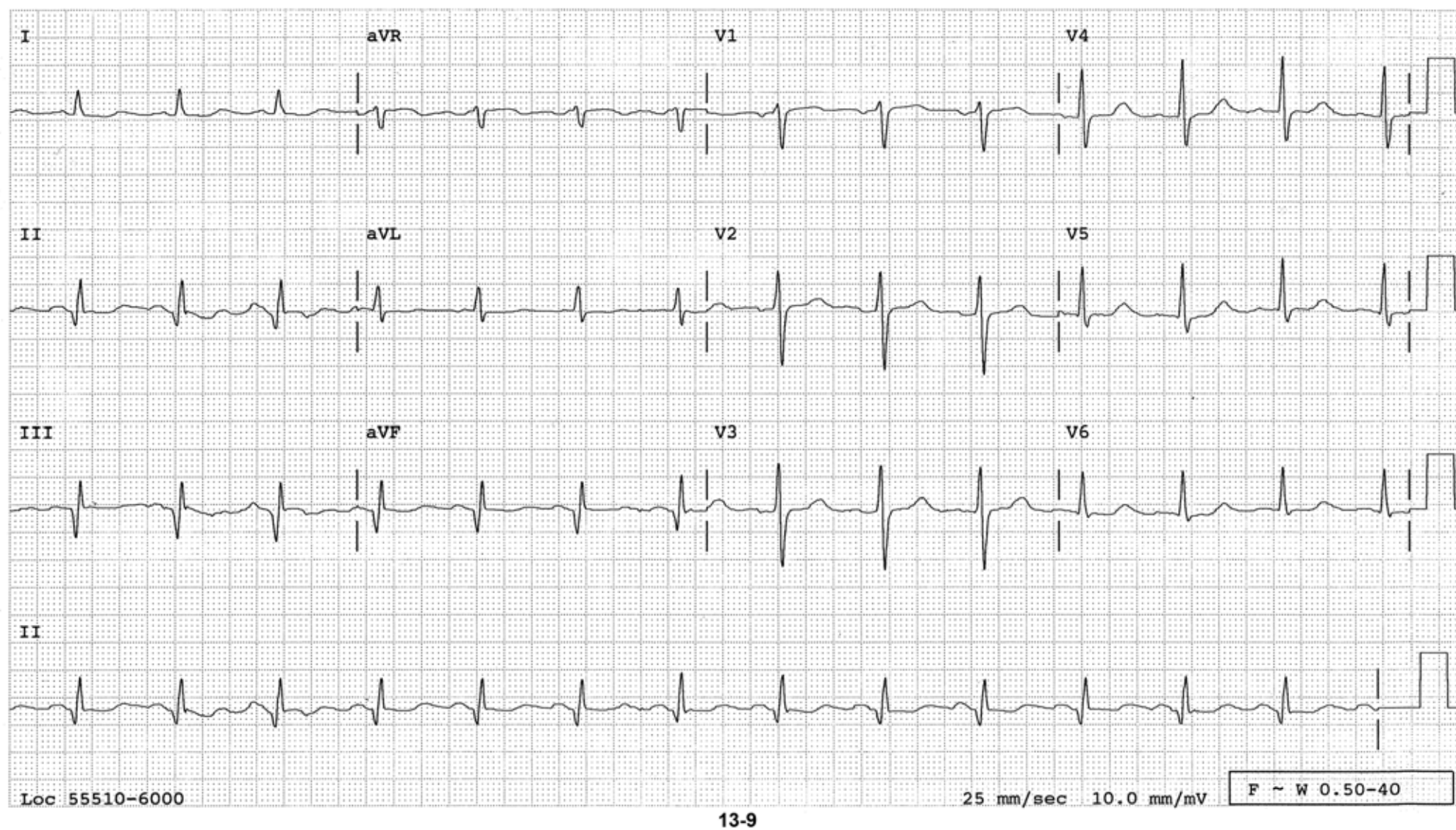
13-7 P waves occur regularly at a rate of about 75/minute. However, only every other P wave results in a QRS indicating 2:1 AV block. Since the QRS is narrow, the location of the block is most likely within the AV node rather than below the His bundle.

Dx: Sinus rhythm with 2:1 AV block



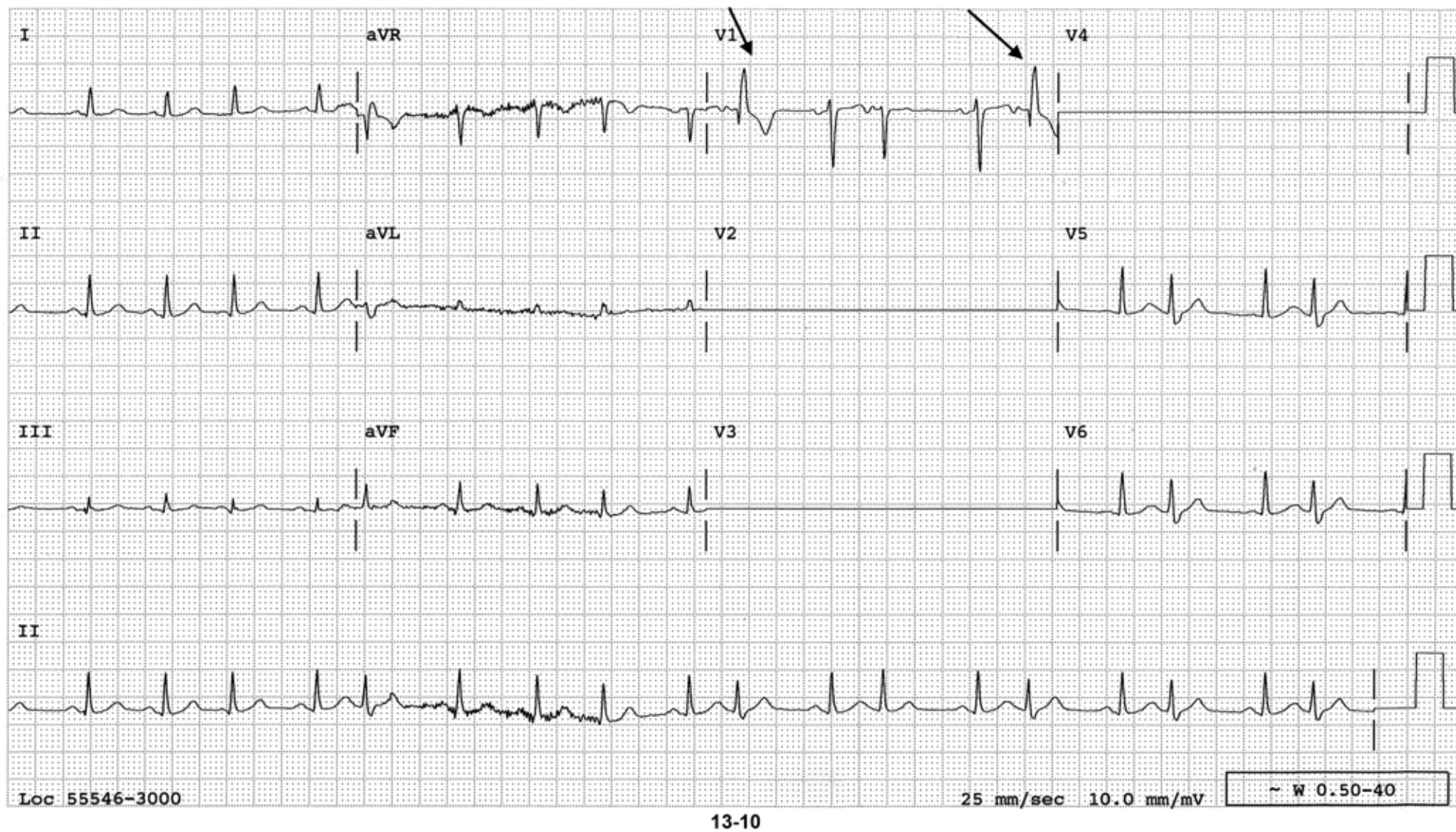
13-8 Narrow QRS tachycardia at a rate of 160/minute. Flutter waves are clearly visible in the inferior leads. Additionally, leads I, aVR, and V₁ reveal that there are two atrial activities for each QRS, further supporting the diagnosis. The ST-segment is largely distorted by the flutter waves and cannot be assessed accurately.

Dx: Atrial flutter with 2:1 AV conduction



13-9 Normal sinus rhythm at a rate of 82/minute. The P waves are mostly negative in V_1 but clearly upright in lead II, indicating LAE. The Q waves are deep and wide enough in the inferior leads indicating old inferior MI.

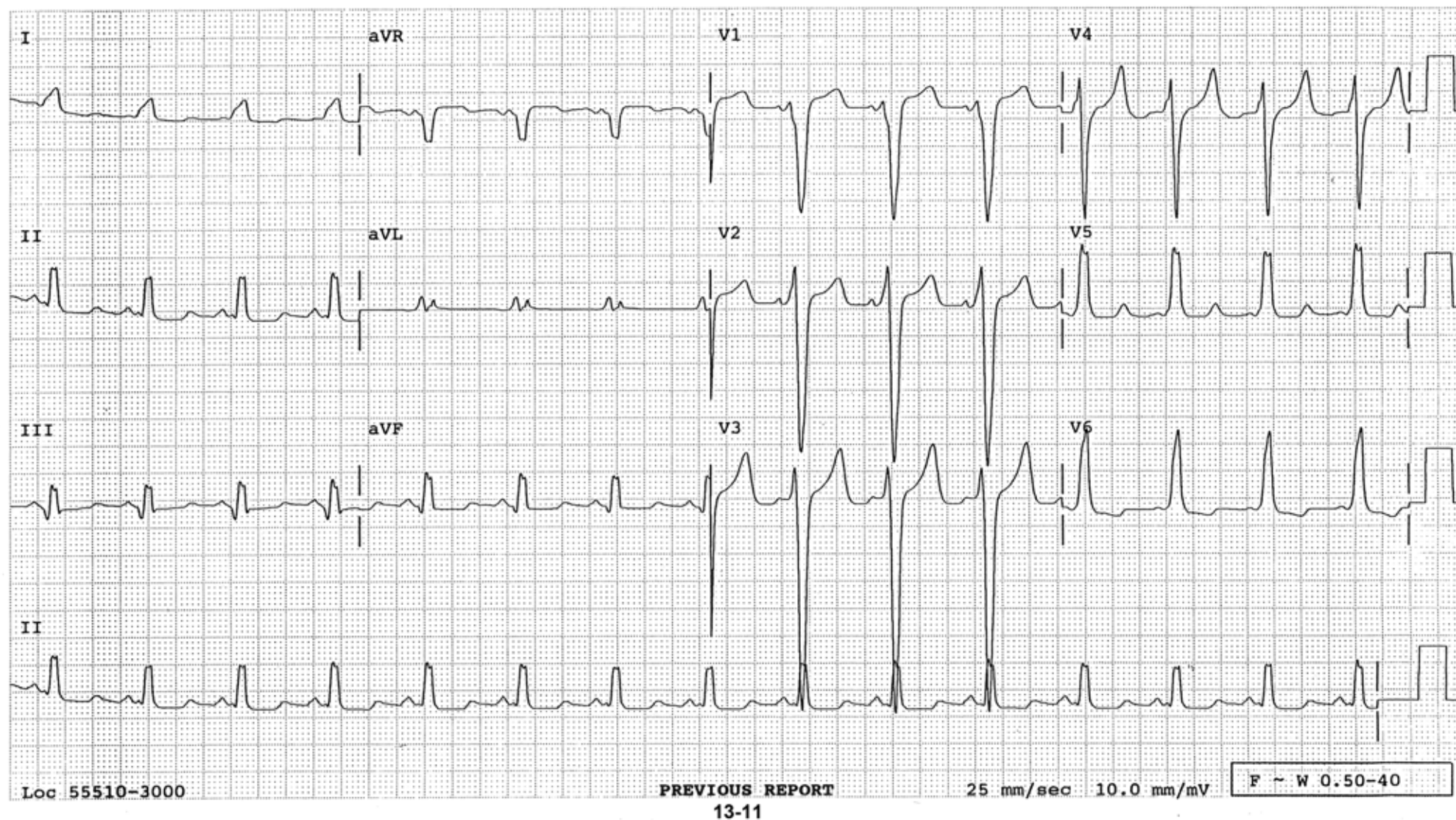
- Dx:
1. NSR
 2. LAE
 3. Old inferior infarct



13-10 Sinus tachycardia at a rate of 115/minute. Many PACs are present. Some of these are aberrantly conducted (↓).

Dx: 1. Sinus tachycardia

2. Multiple atrial premature complexes, some of which are aberrantly conducted



13-11 Sinus rhythm at a rate of 90/minute. The QRS complexes in lead I suggest LBBB. The initial negative deflection in lead III suggests inferior infarct. However, the short P-R interval and delta waves are evident in the precordial leads indicating WPW syndrome. The widening of the QRS in lead I is actually due to the delta wave. The "Q" wave in lead III is actually an inverted delta wave.

Dx: WPW syndrome mimicking either LBBB or inferior infarct

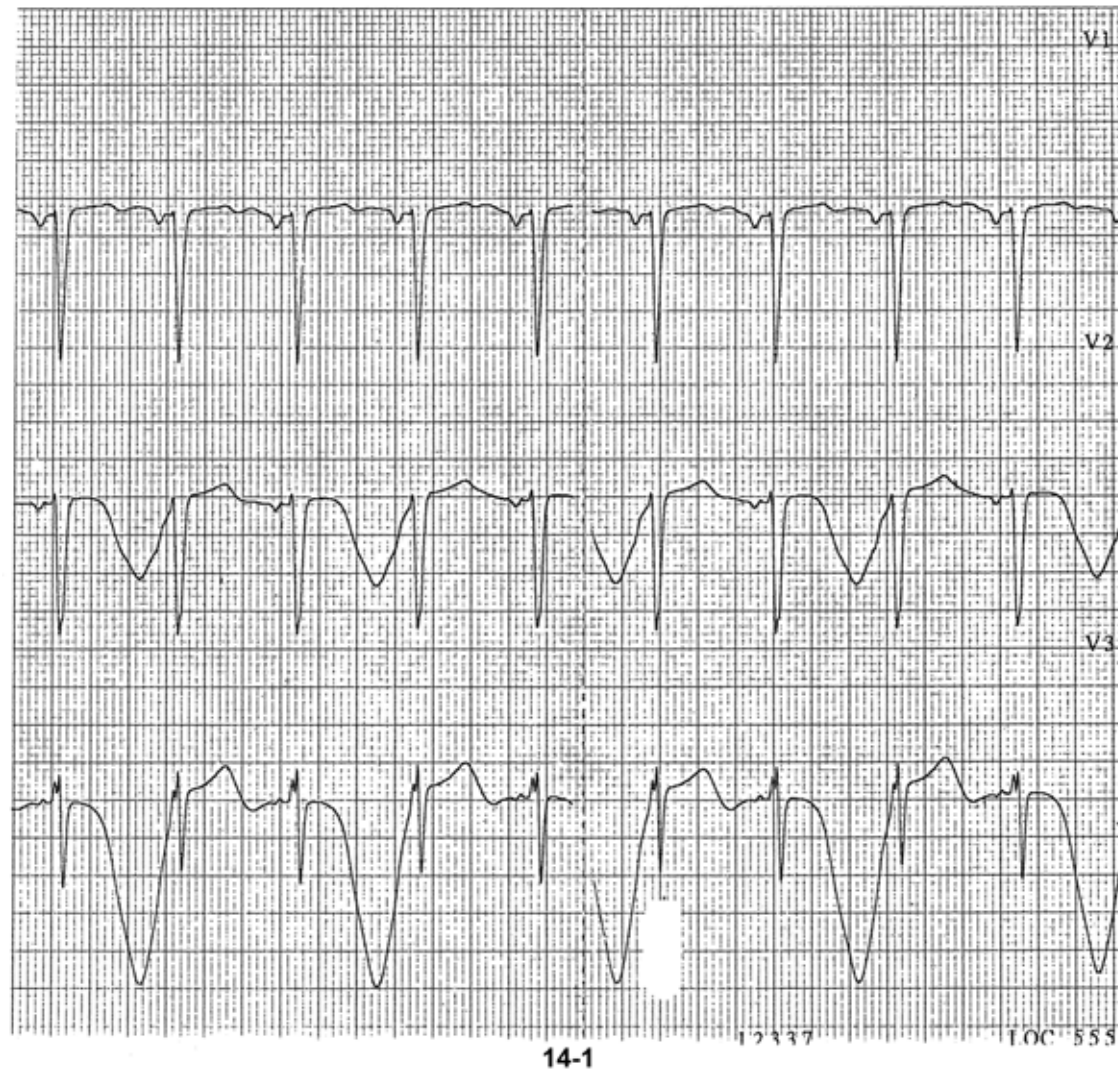


13-12

13-12 A slow regular narrow QRS rhythm is present. Each QRS is preceded by a P wave with a fixed P-R interval suggesting sinus bradycardia. However, there is an additional blip after the T wave which looks good for an extra atrial activity. However, the P-P interval is not regular and these extra P waves occur prematurely. This is an example of nonconducted atrial bigeminy (every other premature P wave is blocked). When there is an unusually slow rhythm, one has to be alerted to the possibility of an extra P wave hidden between the QRSs as in this case.

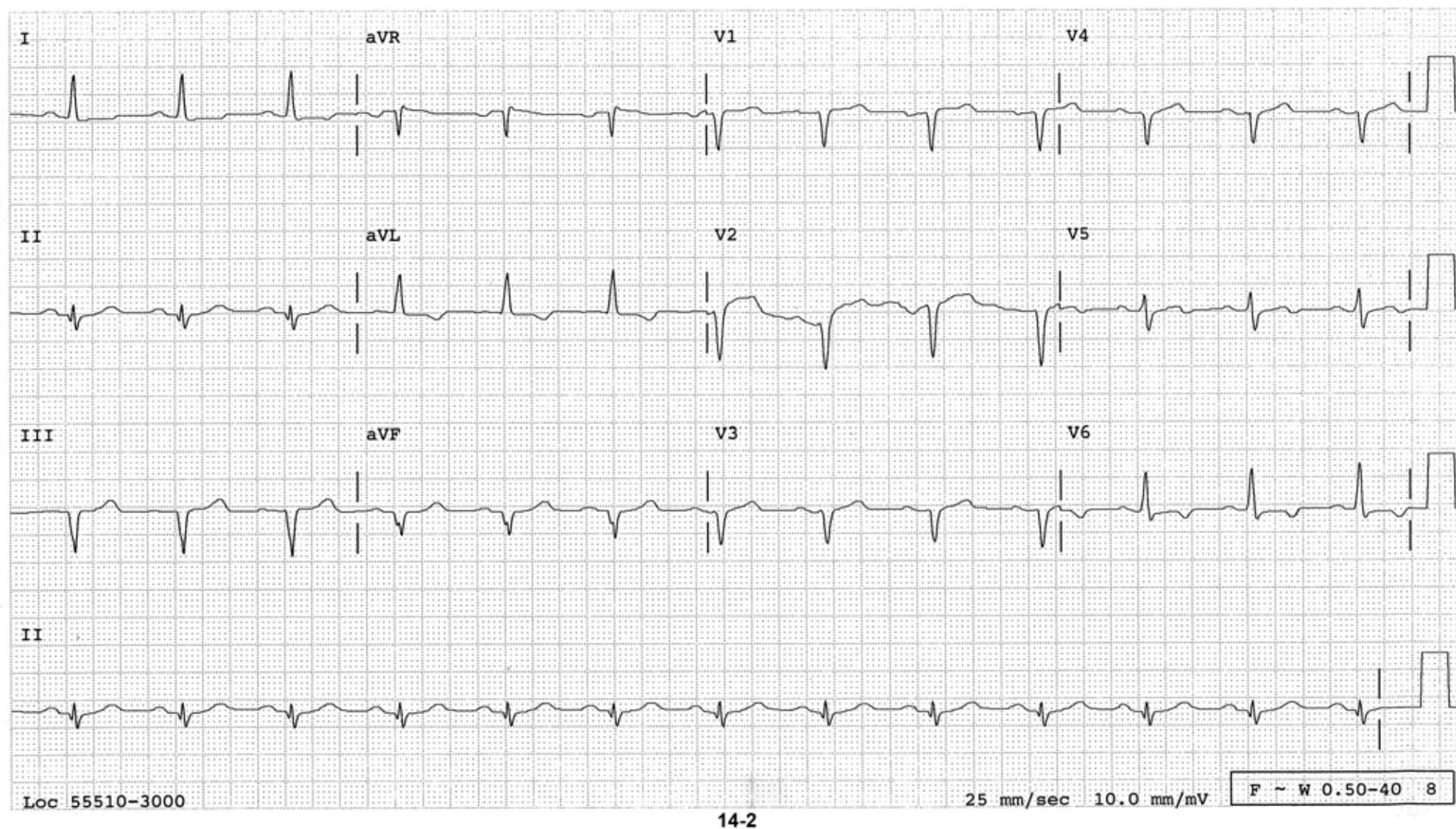
Dx: Sinus rhythm with nonconducted atrial bigeminy

SECTION 14



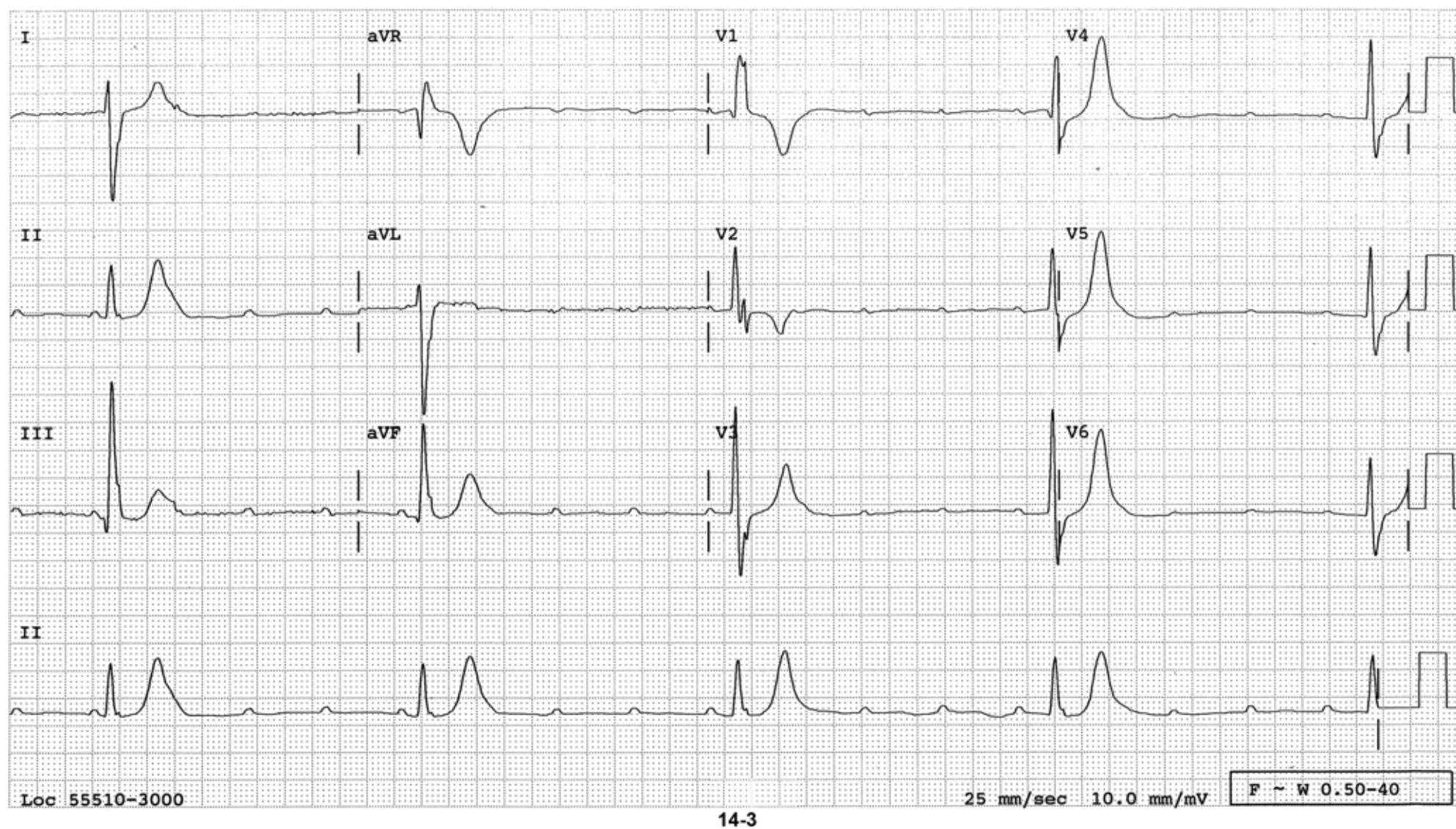
14-1 Normal sinus rhythm at 90/minute. Deeply inverted T wave with a long Q-T interval alternates with a positive T wave (T wave alternans). These findings are often a prelude to *Torsade de Pointes*, and are seen in myocardial ischemia, congenital long QT syndrome, catecholamine surge, or a variety of electrolyte derangements.

- Dx:
1. NSR
 2. T wave alternans, often a prelude to *Torsade de Pointes*



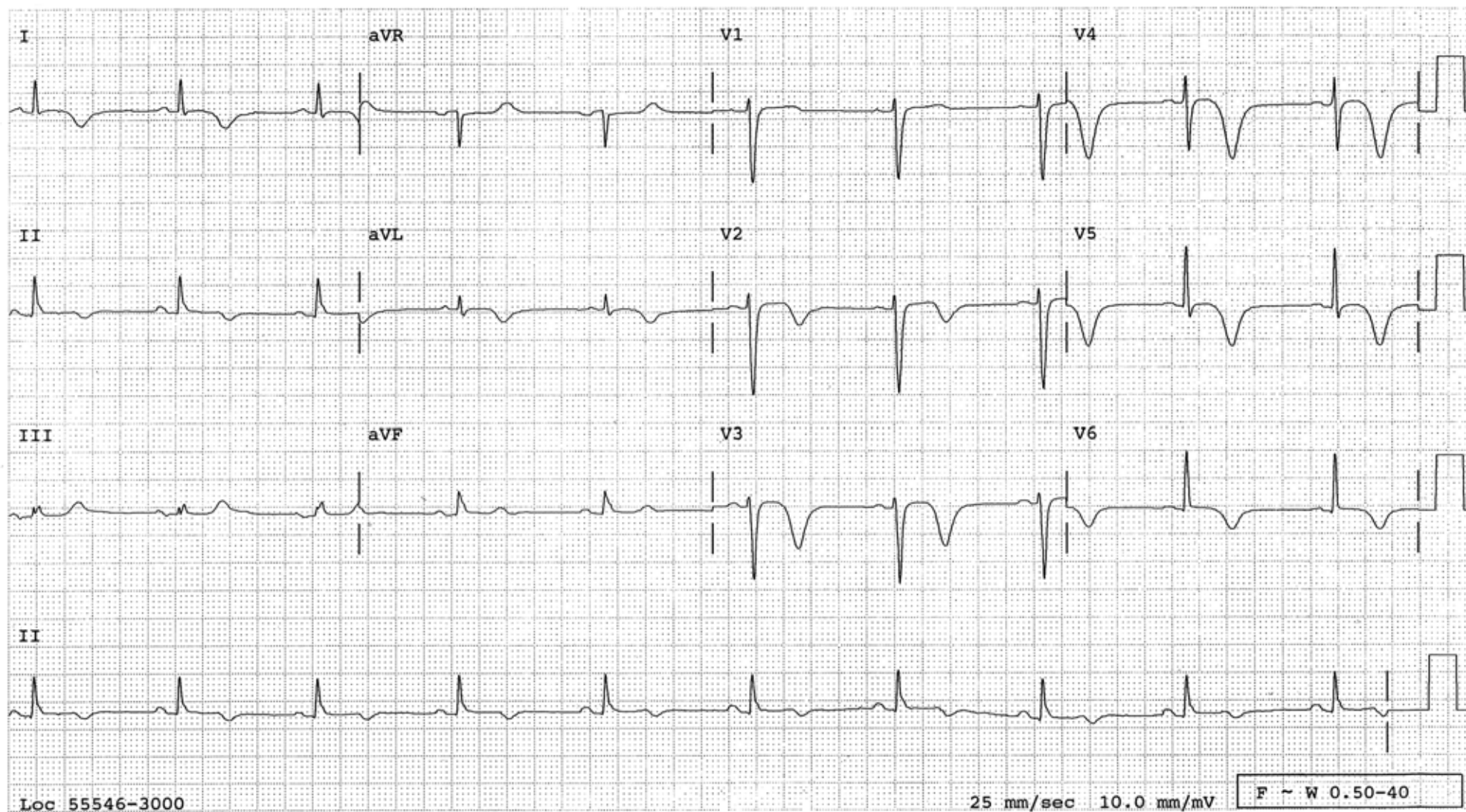
14-2 NSR at a rate of 77/minute. The mean QRS axis is shifted to the left. QS pattern in leads III and aVF may reflect left anterior fascicular block (LAFB) with or without old inferior MI. Lead II holds the key. If it starts with a q wave no matter how small it is, an old inferior MI is present. If it starts with an r wave, an old inferior MI is not present. A QS pattern in leads V_1 - V_4 indicates an old anteroseptal MI as well in this tracing.

- Dx:
1. NSR
 2. LAFB
 3. Old inferior infarct
 4. Old anteroseptal infarct



14-3 Wide QRSs are occurring regularly at a rate of 26/minute. P waves occur regularly at 110/minute and have no fixed relationship to the QRS indicating complete AV block. The QRSs are wide and may reflect RBBB and posterior fascicular block (extreme Rt axis deviation). T waves are tall, pointed and symmetric. These findings are highly suggestive of hyperkalemia. In that case, the correction of hyperkalemia may reverse the complete AV block.

Dx: 3° AV block, which may be caused by hyperkalemia in a patient with bifascicular block (RBBB and posterior fascicular block)

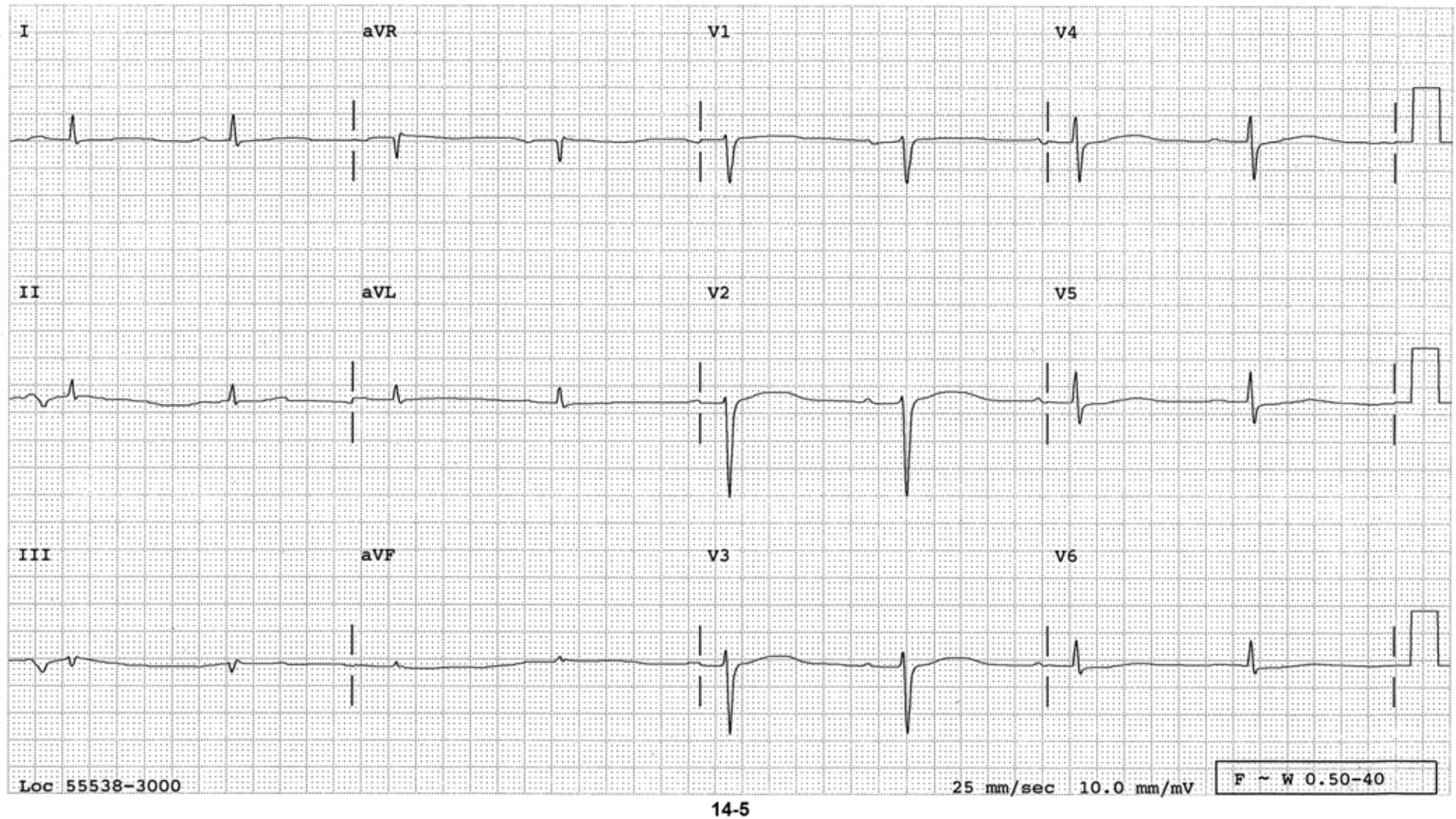


14-4

14-4 NSR at a rate of 58/minute. The T waves are markedly inverted symmetrically with a long Q-T interval in the precordial leads suggesting either subendocardial ischemia or infarction. These changes are also consistent with the so-called stress cardiomyopathy which is seen in conditions such as CNS events or other stressful situations with catecholamine surge.

Dx: 1. NSR

2. T wave changes suggestive of subendocardial ischemia or infarction, or stress cardiomyopathy



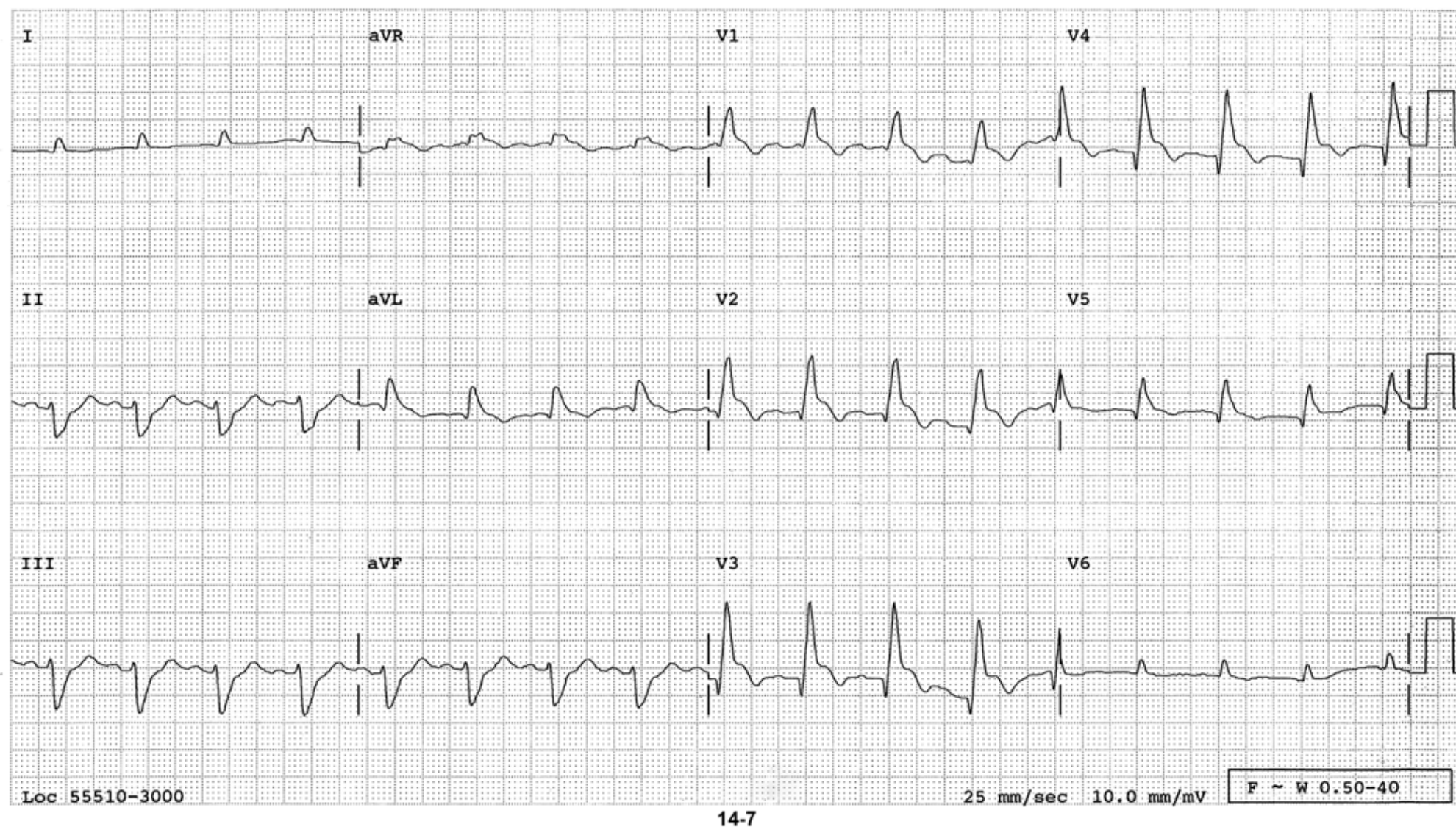
14-5 Sinus bradycardia at a rate of 49/minute. The P-R interval is prolonged to 280 milliseconds. The striking features of this tracing are the flat T waves with a markedly prolonged Q-T interval. The long Q-T interval could be secondary to medications, inhomogeneous sympathetic influence to the heart, other metabolic derangements, or congenital. This patient is at risk of developing *Torsade de Pointes*.

- Dx:
1. Sinus bradycardia
 2. 1° AV block
 3. T wave flattening
 4. Markedly prolonged Q-T interval



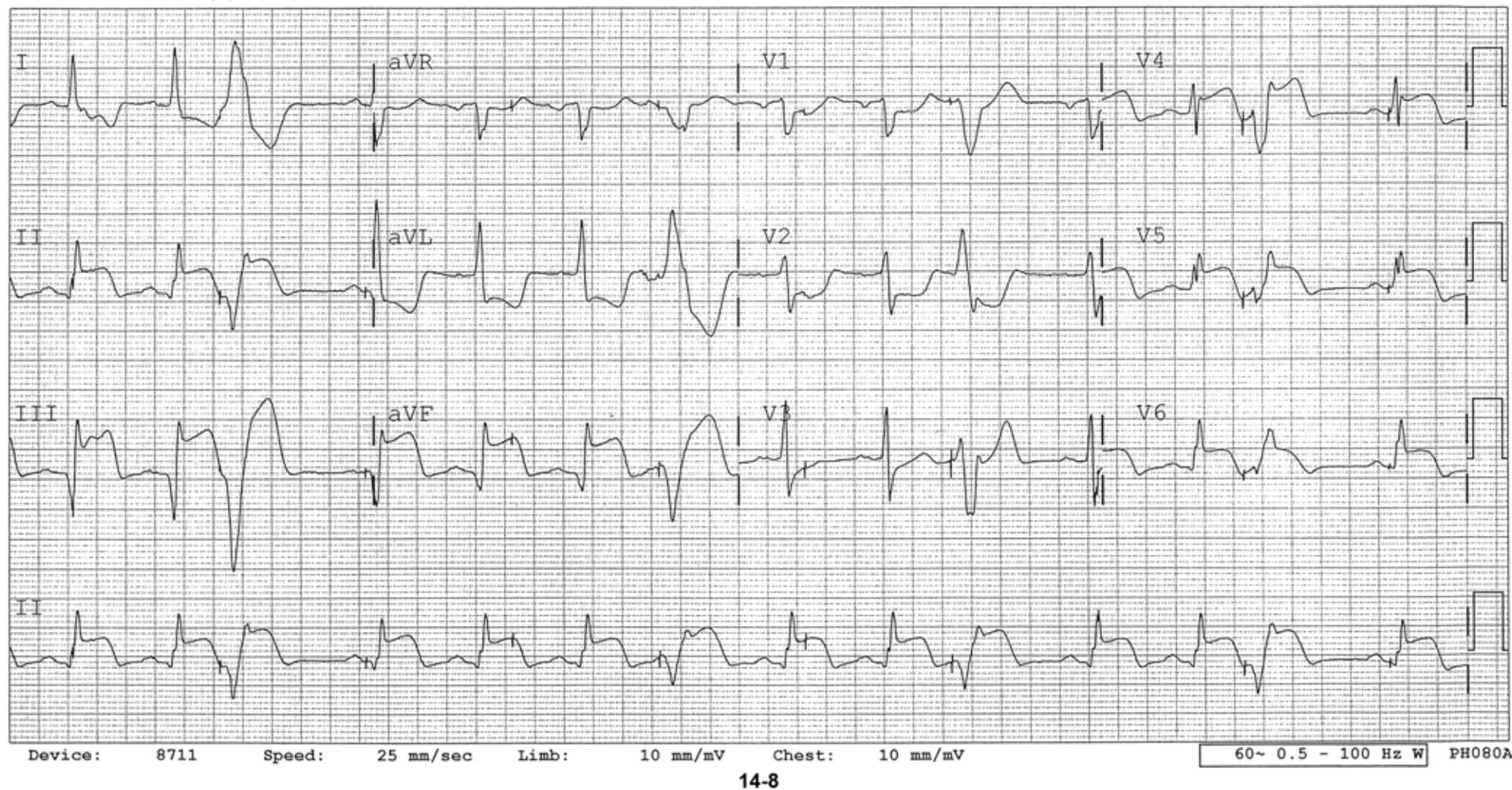
14-6 Irregularly irregular rhythm at a rate of 88/minute. The QRSs are markedly widened. The T waves of the second QRS complex in V₄-V₆ are tall, pointed, and narrow, suggesting hyperkalemia. The ST elevation in leads VI and III is from hyperkalemia (pseudoinfarction pattern, or acquired Brugada ECG pattern).

- Dx:
1. Atrial fibrillation
 2. Findings consistent with hyperkalemia



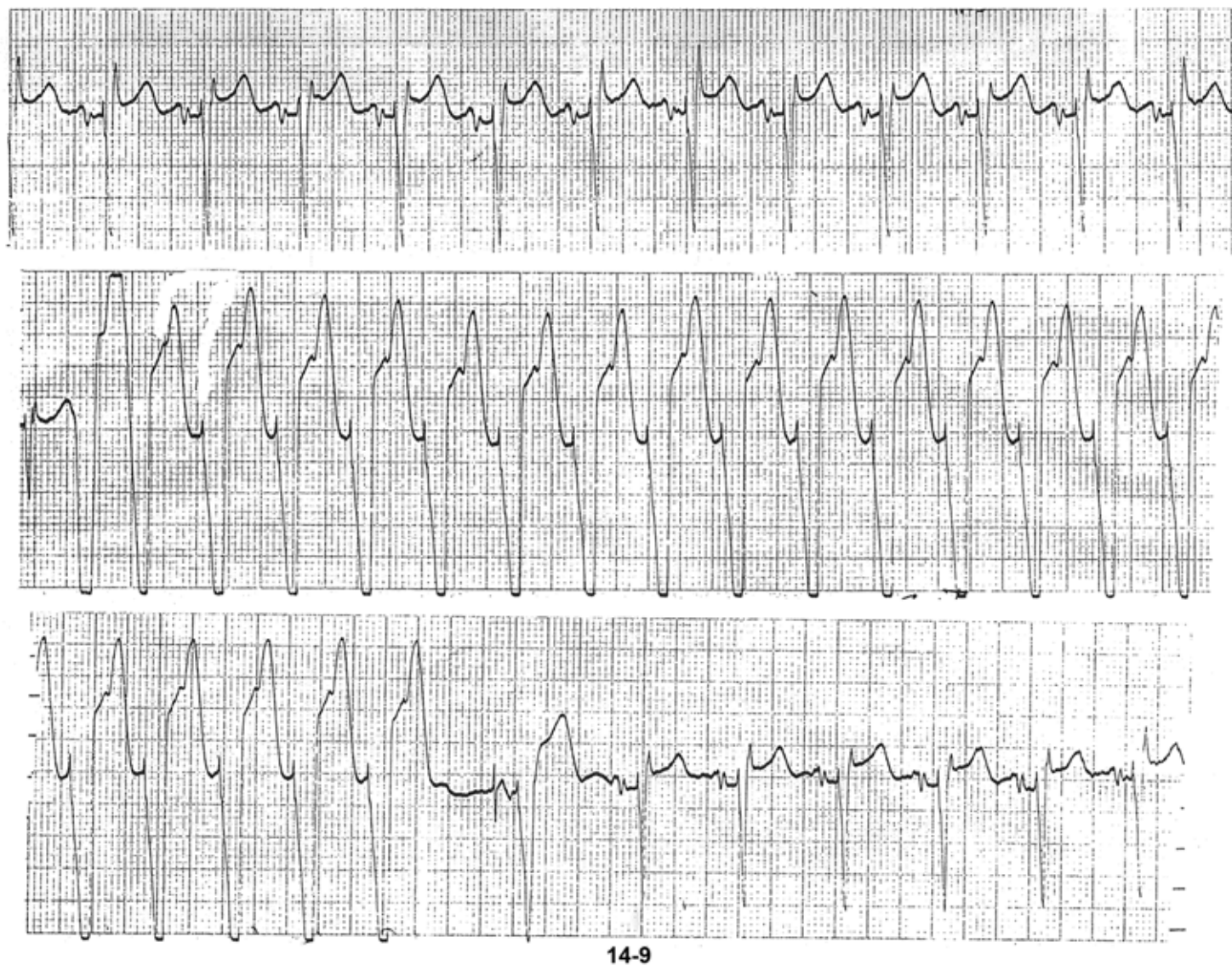
14-7 Normal sinus rhythm at a rate of 99/minute. Complete RBBB and LAFB make BIFB. The initial r wave (the first "rabbit ear") of the rsR' is not present due to AMI. Since the ST-segment is elevated in these leads, the infarction is acute.

- Dx:
1. NSR
 2. Bifascicular block consisting of RBBB and LAFB
 3. Acute anterior infarct



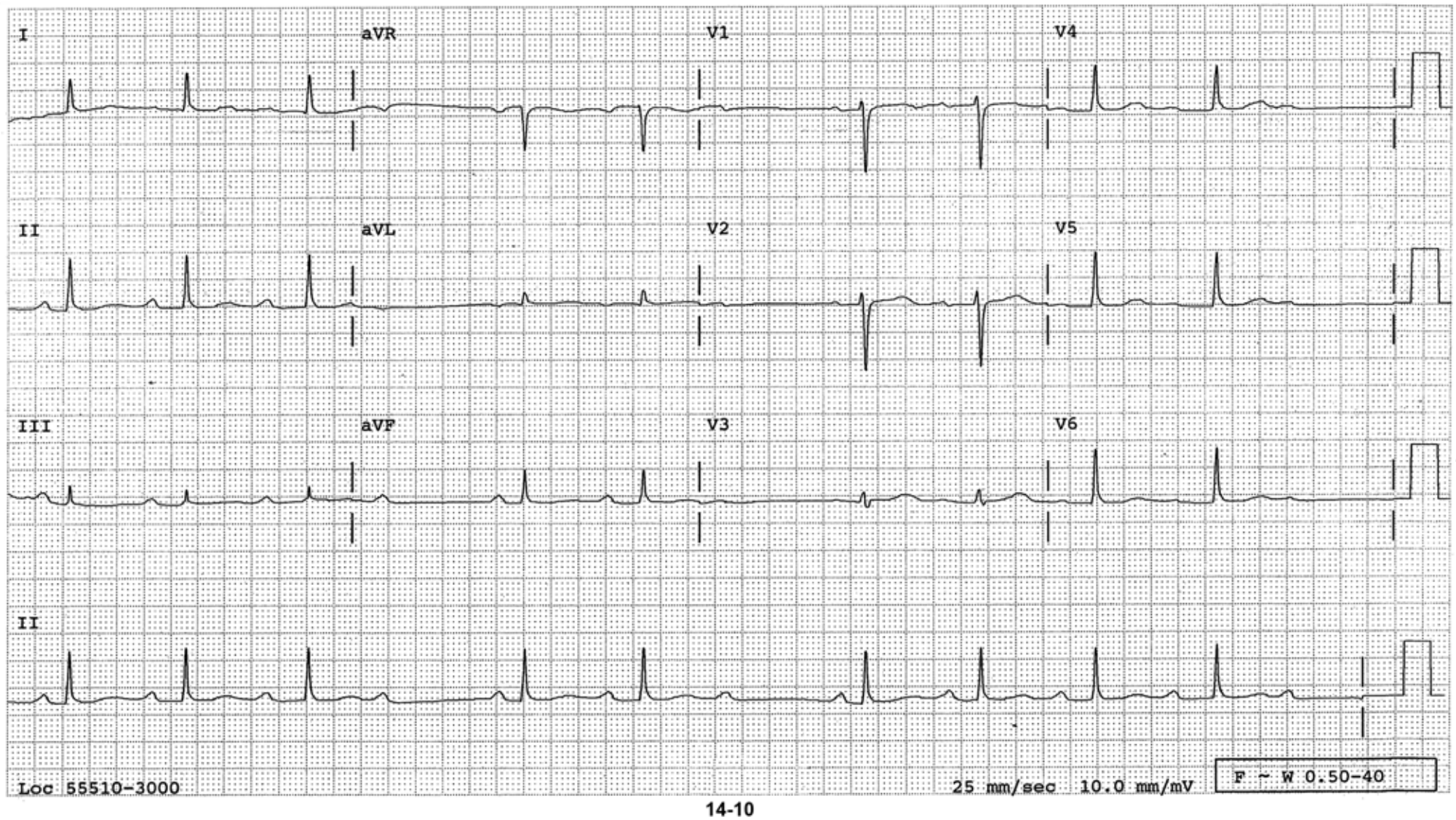
14-8 Normal sinus rhythm at 80/minute. Inferoposterolateral STEMI is evident. The patient has a pacemaker in the ventricle which fires regularly in a fixed mode. If the pacer-spike occurs outside of the ventricular refractory period, it will capture the ventricle (third, seventh, tenth and thirteenth complexes). These paced QRSs also reveal the STEMI very effectively. Thus, when one is faced with a patient for possible MI and the rhythm is entirely paced, one should not say the ECG is useless because, at times, it may still show the infarction very effectively.

- Dx:
1. NSR, fixed mode ventricular pacer spikes with occasional capture
 2. STEMI of inferoposterolateral wall, which is also revealed in the paced complexes



- 14-9 This is an example of a pacemaker-mediated tachycardia. This patient has an AV sequential pacemaker. The first two wide complexes in the middle strip are PVCs. With the second PVC, there is a retrograde P wave which is sensed by the atrial lead and it commands the ventricular pacemaker to fire and capture the ventricles, which again causes a retrograde P wave which is sensed by the atrial lead and the cycle repeats again and again, hence called “endless loop tachycardia” or “pacemaker-mediated tachycardia”. The most effective way to terminate the tachycardia is applying a magnet over the pulse generator which will disable the atrial sensing mechanism, hence no more sensing of the retrograde P wave. The prevention of this tachycardia is achieved by lengthening the refractory period of the atrial lead so that the retrograde P wave will not be sensed. Note the atrial pacemaker spike in the first beat after the conversion.

Dx: Pacemaker-mediated tachycardia



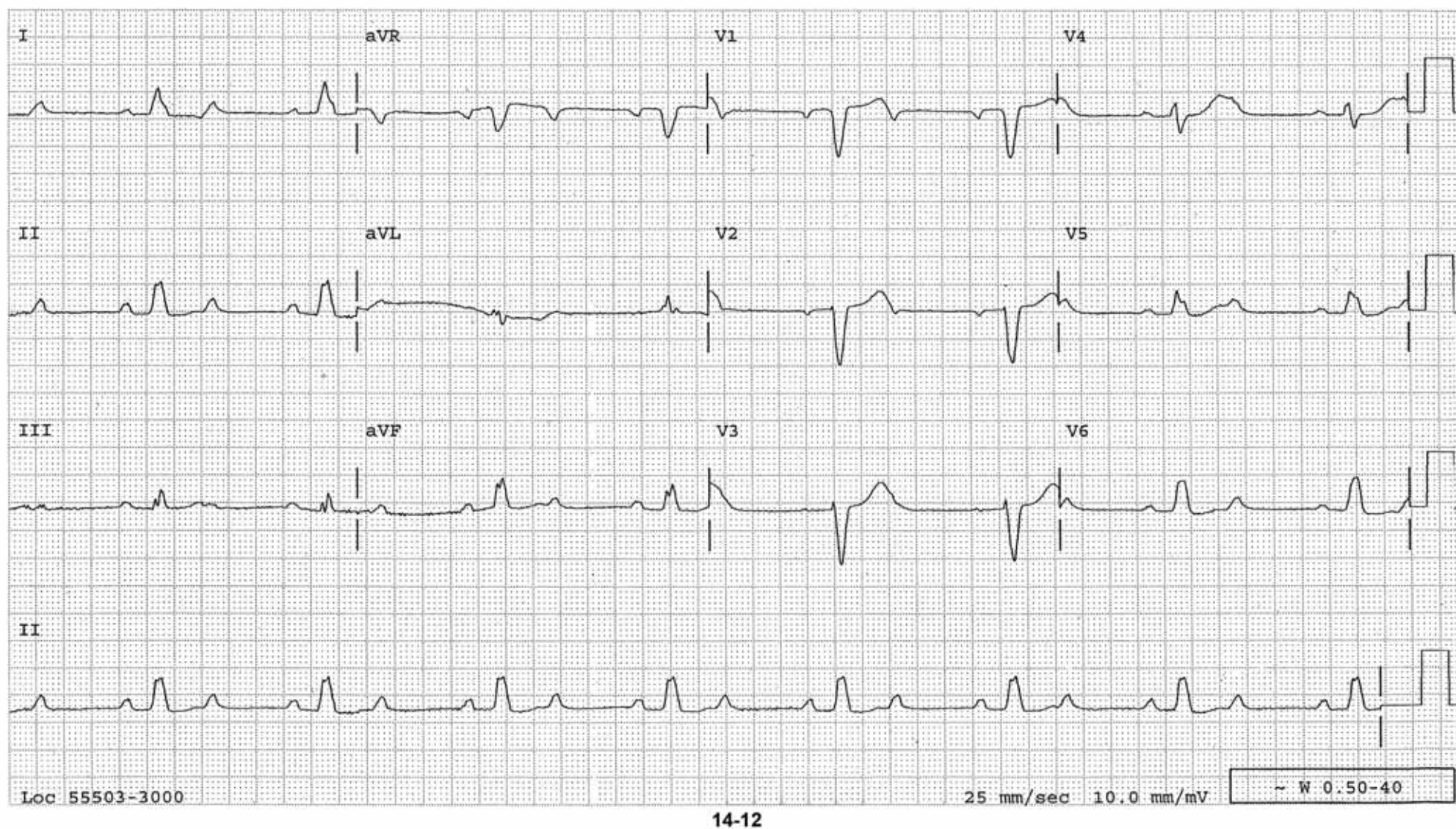
14-10 Sinus rhythm at a rate of 69/minute. Some P waves are not conducted to the ventricle. Prior to these, the P-R interval progressively lengthens; a typical Type I 2° AV block.

Dx: NSR with Type I 2° AV block



14-11 Sinus tachycardia at a rate of 118/minute. The P wave is tall measuring about 6 mm in lead II and indicates RAE. There are no other features of RVH such as RAD, tall R waves in the right precordial leads, or S waves in the left precordial leads.

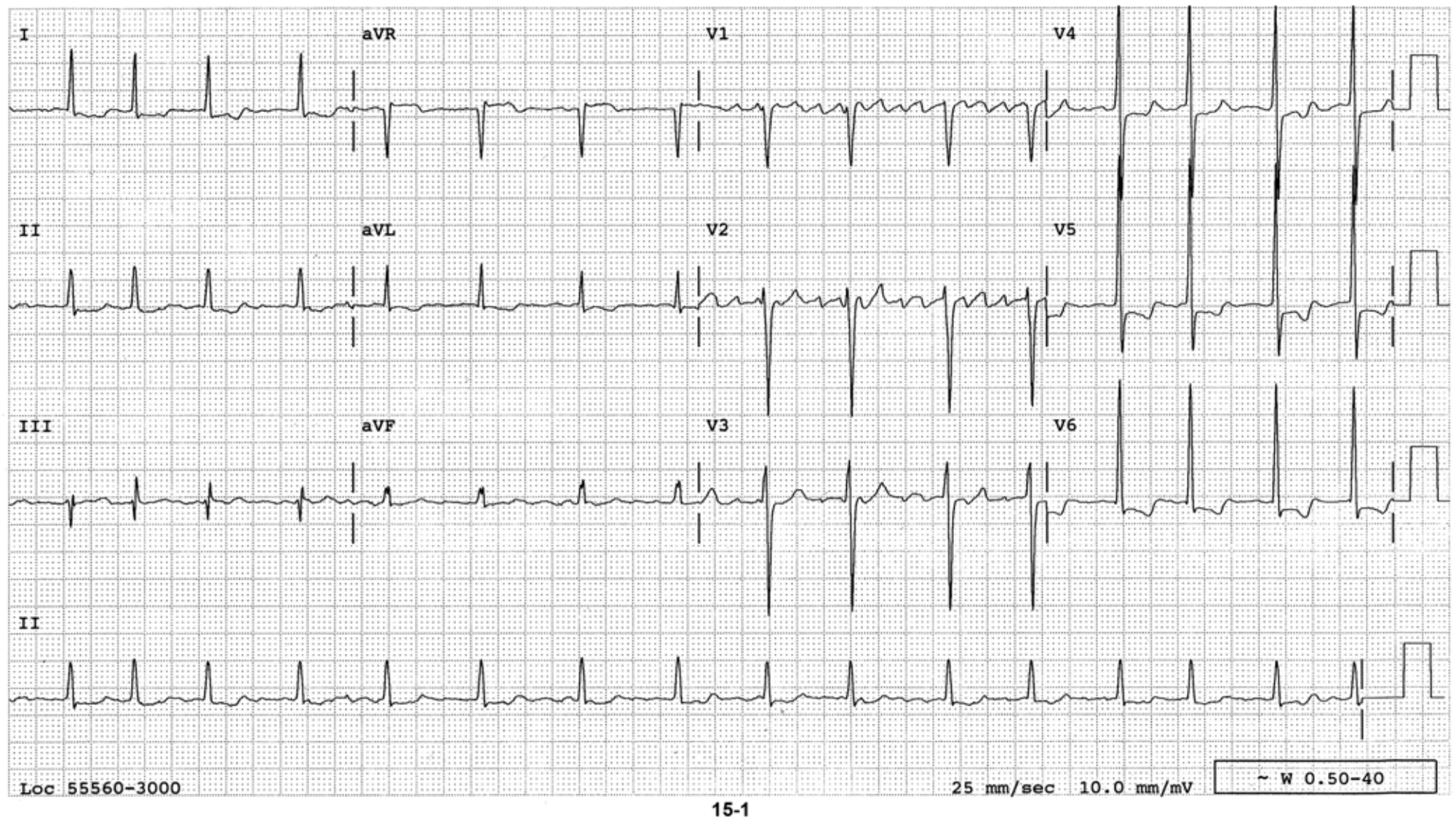
- Dx: 1. Sinus tachycardia
2. RAE



14-12 Regular wide QRS rhythm at 50/minute. There are two P waves between the QRSs with a fixed P-R interval indicating 2:1 AV block. Monophasic R waves in leads I, aVL and V₆, and almost entirely negative QRSs in V₁-V₃ are typical of LBBB.

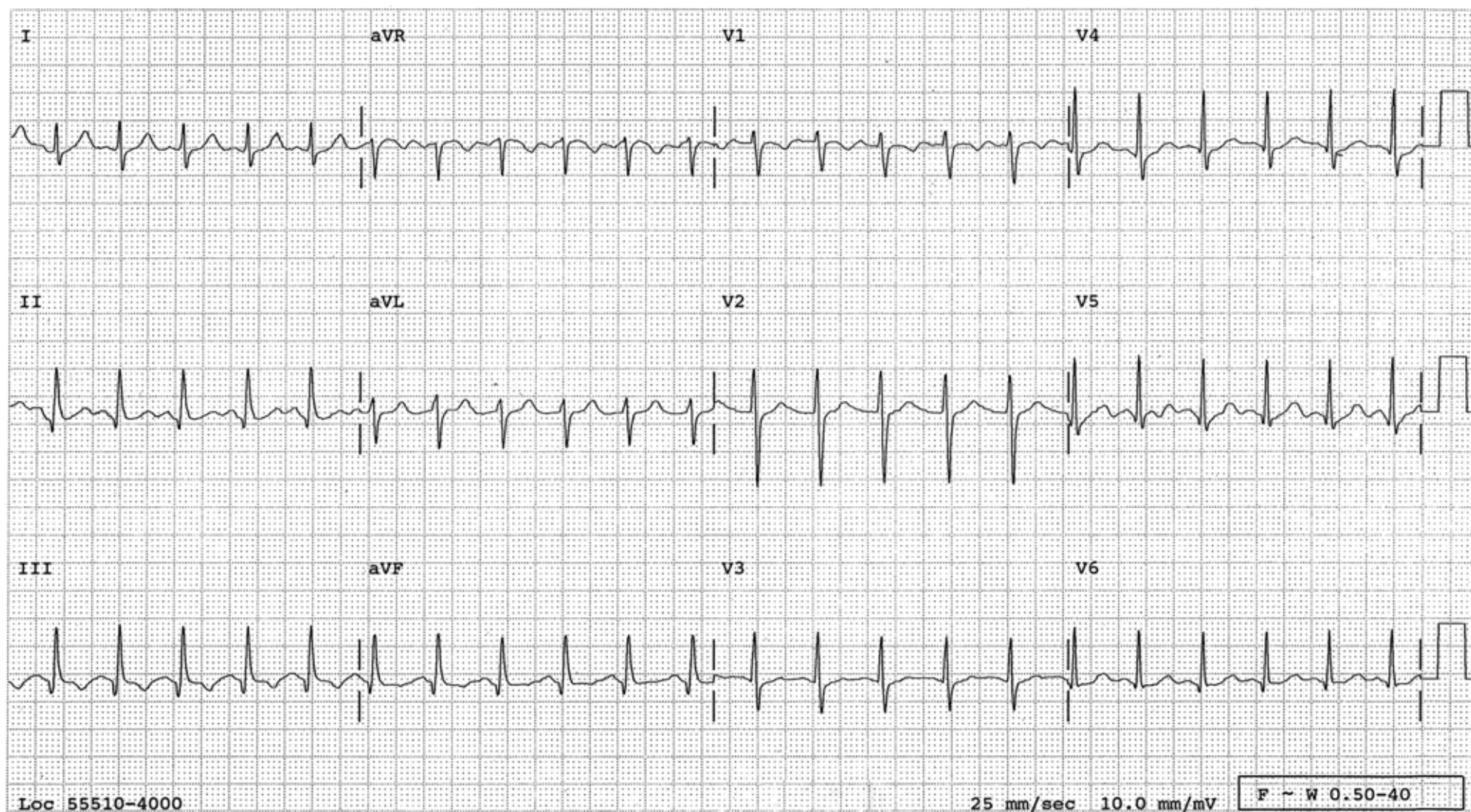
- Dx:
1. Sinus rhythm with 2:1 AV block
 2. LBBB

SECTION 15



15-1 Atrial fibrillation with a ventricular response of 97/minute. Voltage criteria and ST-T changes for LVH are present. The fibrillatory waves are best seen in V_1 which is usually the case.

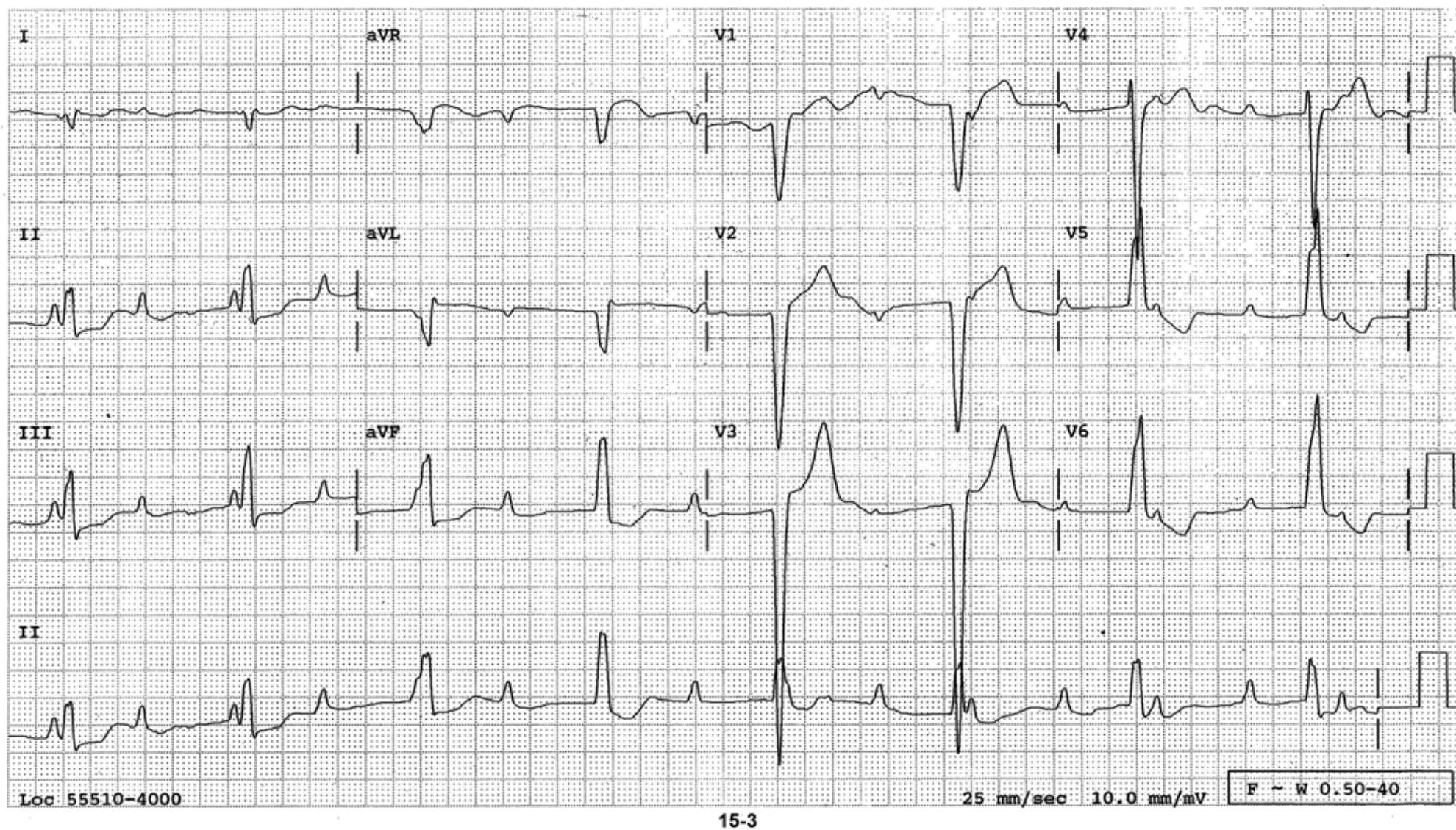
- Dx: 1. Atrial fibrillation with a ventricular response of 97/minute
2. LVH



15-2

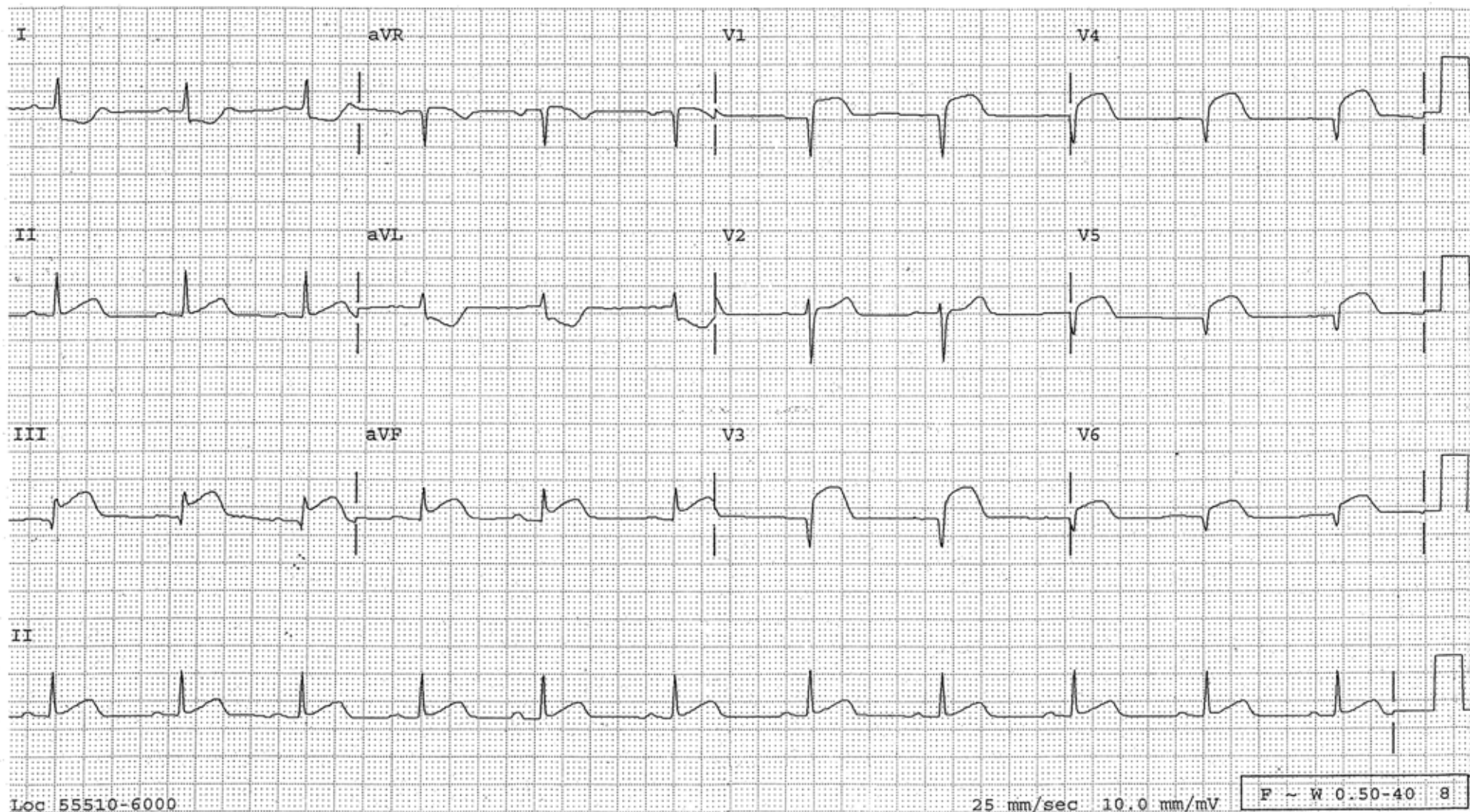
15-2 Sinus tachycardia at a rate of 131/minute. There is an S wave in lead I and a Q wave with T wave inversion in lead III, making a typical $S_1Q_3T_3$ pattern. This pattern combined with sinus tachycardia is strongly suggestive of pulmonary embolism. The same pattern seen in a patient with a slower HR is less likely to be associated with pulmonary embolism. A large enough pulmonary embolism to cause these ECG changes should be associated with a significant degree of hypoxia which should accompany a sinus tachycardia.

- Dx:
1. Sinus tachycardia
 2. $S_1Q_3T_3$ pattern, highly suggestive of pulmonary embolism



15-3 Wide QRSs occur regularly at 47/minute. The P waves occur regularly on its own at a rate of 90/minute and these P waves have no relationship to the QRSs indicating complete AV block. Since the QRS is wide and the rate is slow, the escape mechanism must be originating from the ventricle rather than from the AV junction. The P wave is taller than 2.5 mm in lead II indicating RAE.

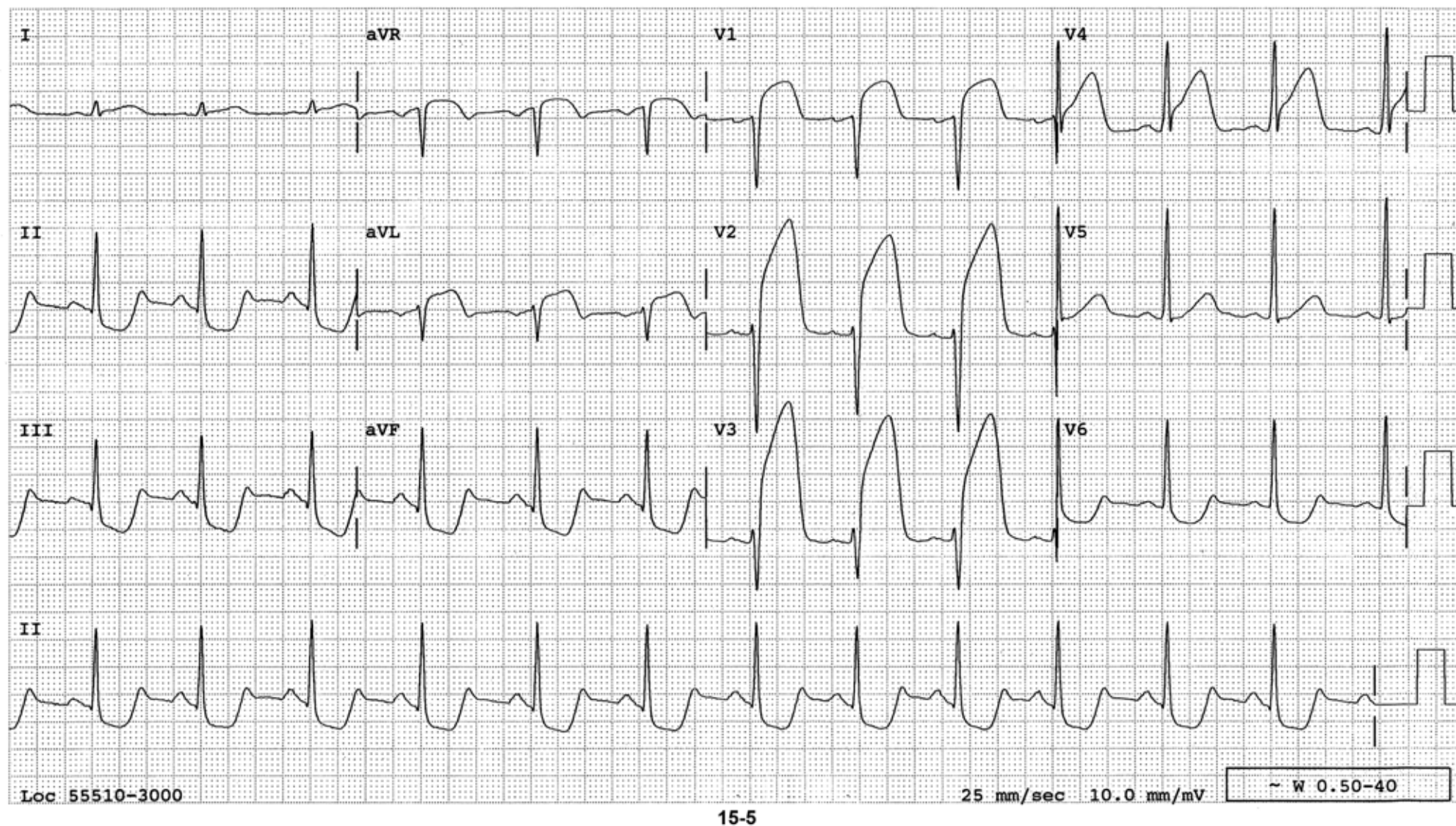
- Dx: 1. Complete AV block with ventricular escape rhythm
2. RAE



15-4

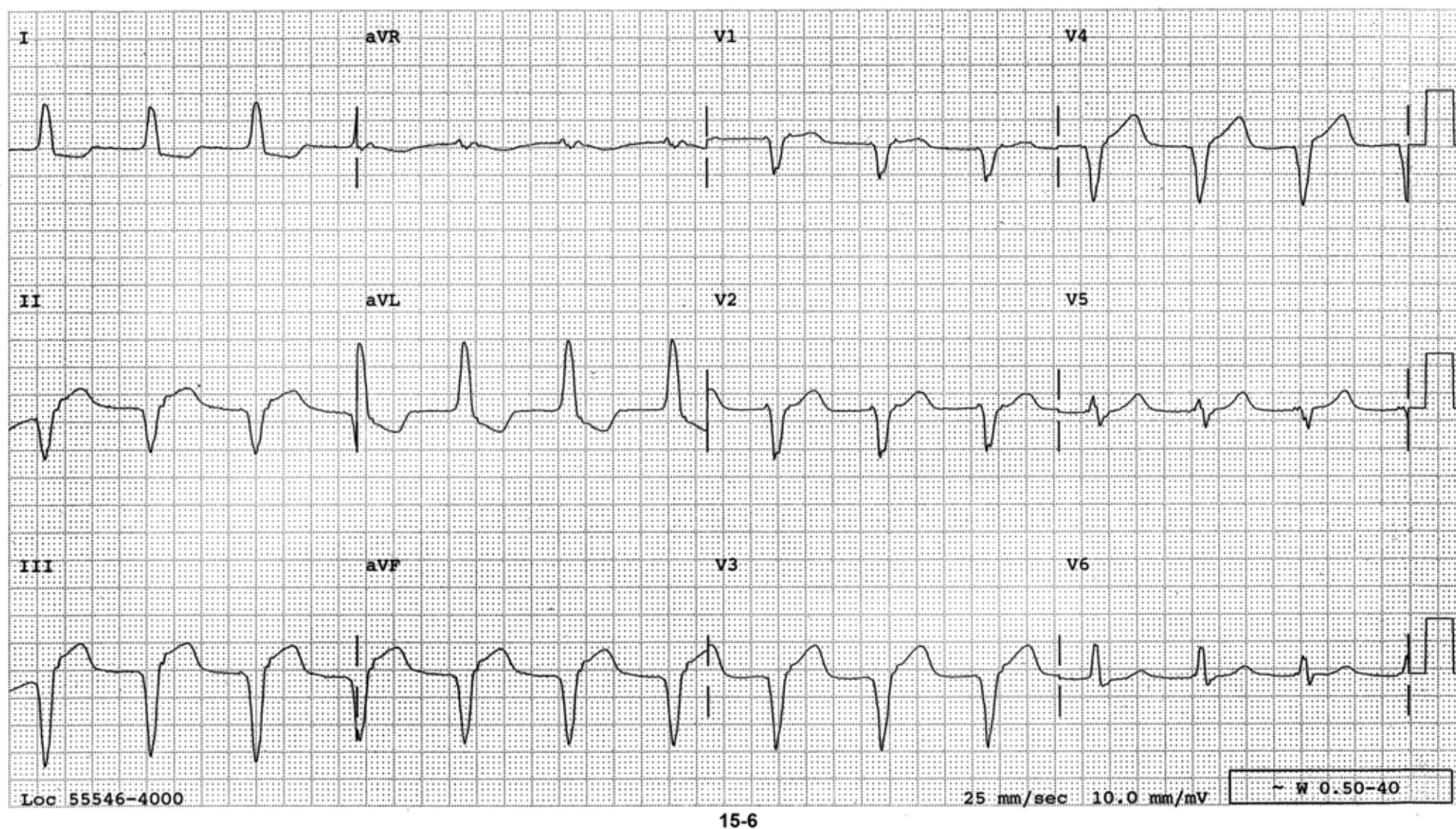
15-4 Inferior infarct is evident (ST elevation in III and aVF). ST-segment is elevated in the precordial leads raising the possibility of anterior wall infarct as well. But actually, the precordial leads are right-sided and reflect RV infarct. The clue that these are right-sided precordial leads is that the QRS vector in V_6 is opposite from QRS vectors of leads I and aVL. In left-sided precordial leads, the QRSs in leads I, aVL, and V_6 should look similar because these leads face the heart from similar angles. In the right-sided precordial leads, it is not the Q waves but ST elevation that indicates RV infarct. Q waves are normally present in right precordial leads. RV involvement is already predicted from the ST depression in lead I in addition to lead aVL.

Dx: Acute inferior infarct with RV involvement



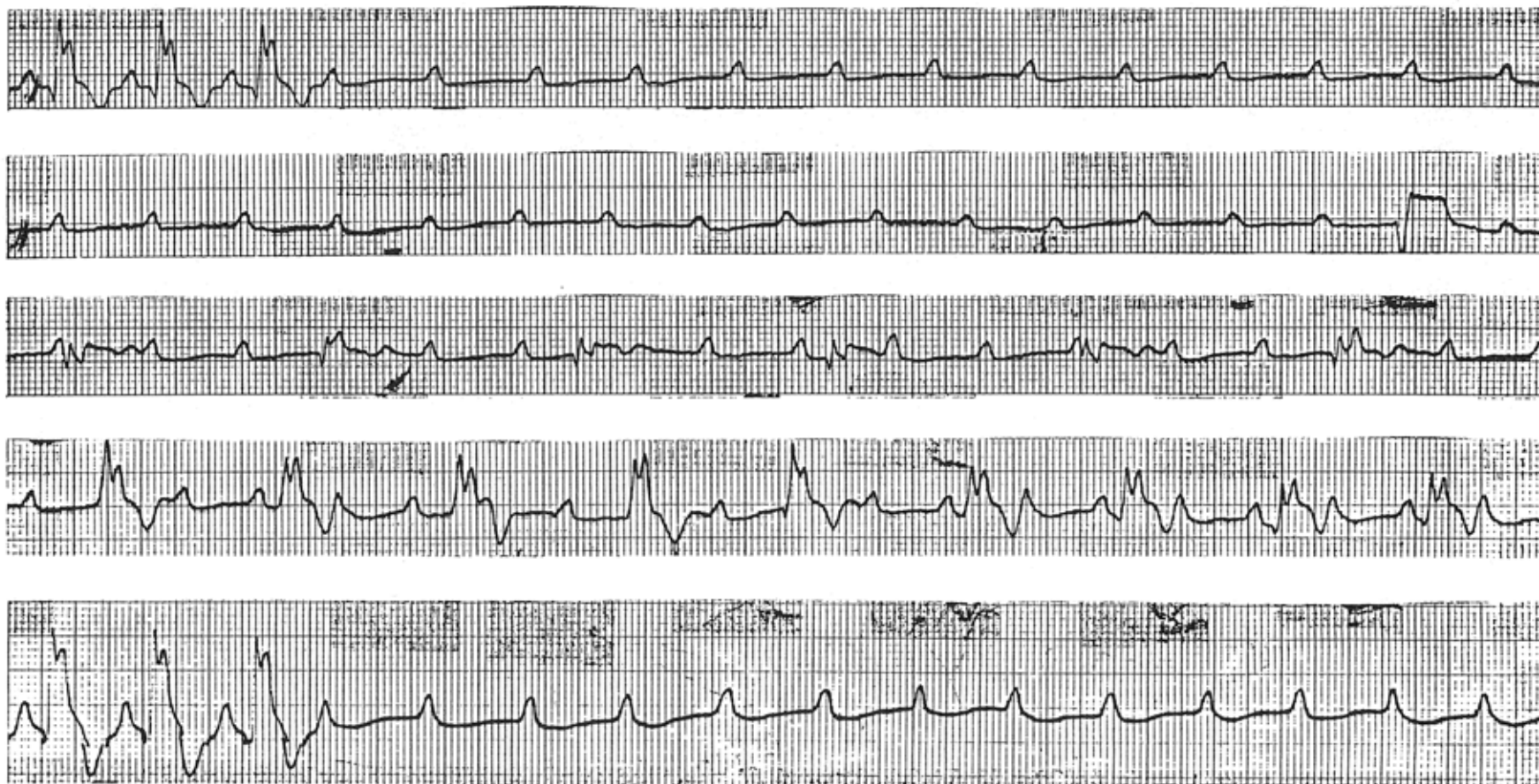
15-5 Normal sinus rhythm at a rate of 77/minute. Marked ST elevation in the precordial leads and leads I and aVL indicate acute anterior MI. The ST-T depression in the inferior leads is reciprocal changes of ST elevation in aVL, and there is nothing wrong with the inferior wall. Involvement of aVL in anterior MI indicates proximal left anterior descending (LAD) occlusion since aVL represents high lateral wall, which is perfused by first diagonal branch, which in turn takes off from proximal LAD. In anterior MI, reciprocal ST depressions in inferior leads occur only if aVL is involved in the infarction process.

- Dx: 1. NSR
2. Acute anterior infarct due to proximal LAD occlusion



15-6 Wide QRSs occur regularly at a rate of 80/minute. No P waves are recognizable in front of the QRS. However, the upstroke of the T waves is deformed in many leads highly suggestive of retrograde P wave. This is an example of accelerated idioventricular rhythm with 1:1 VA conduction.

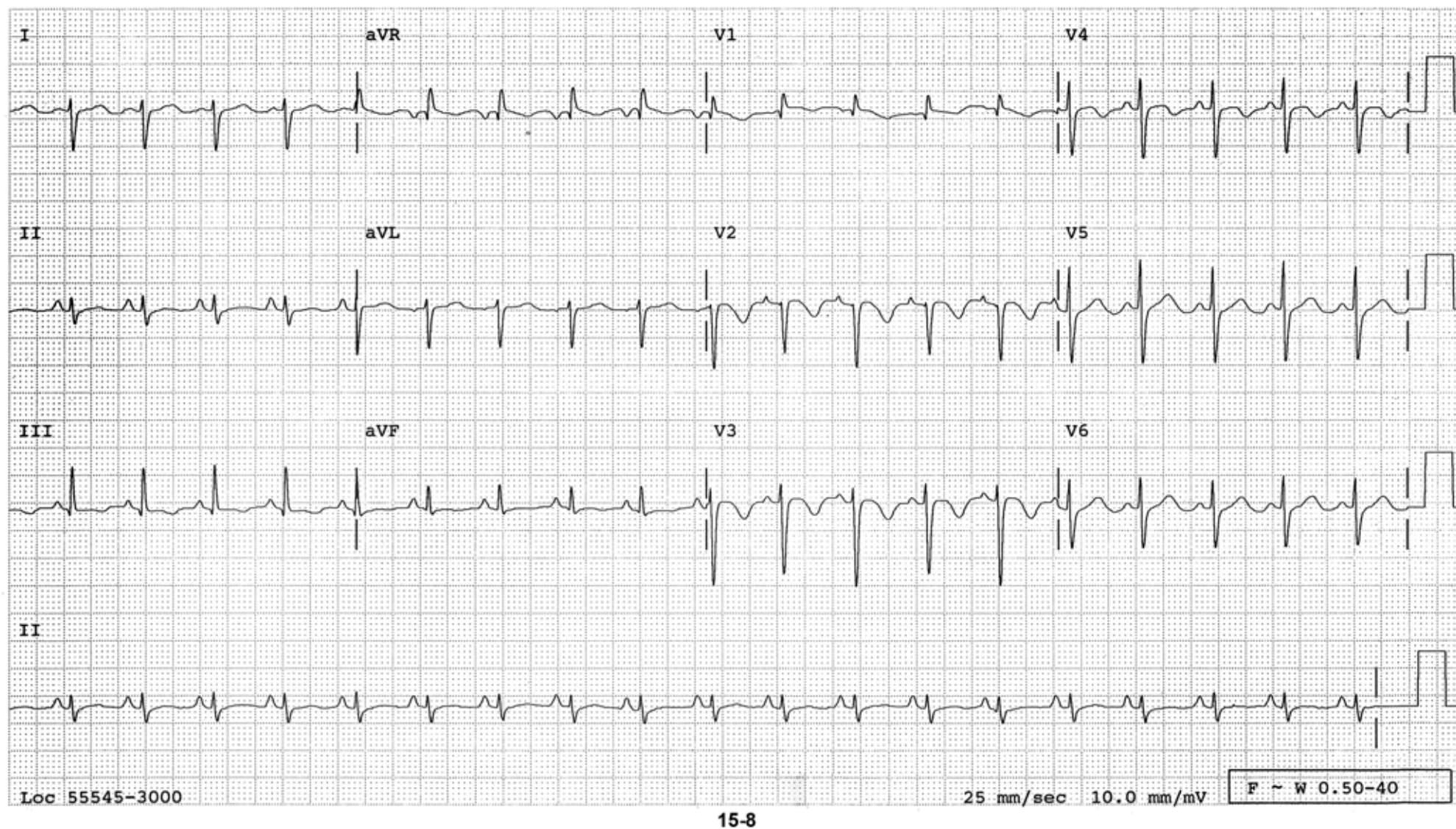
Dx: Accelerated idioventricular rhythm with 1:1 VA conduction



15-7

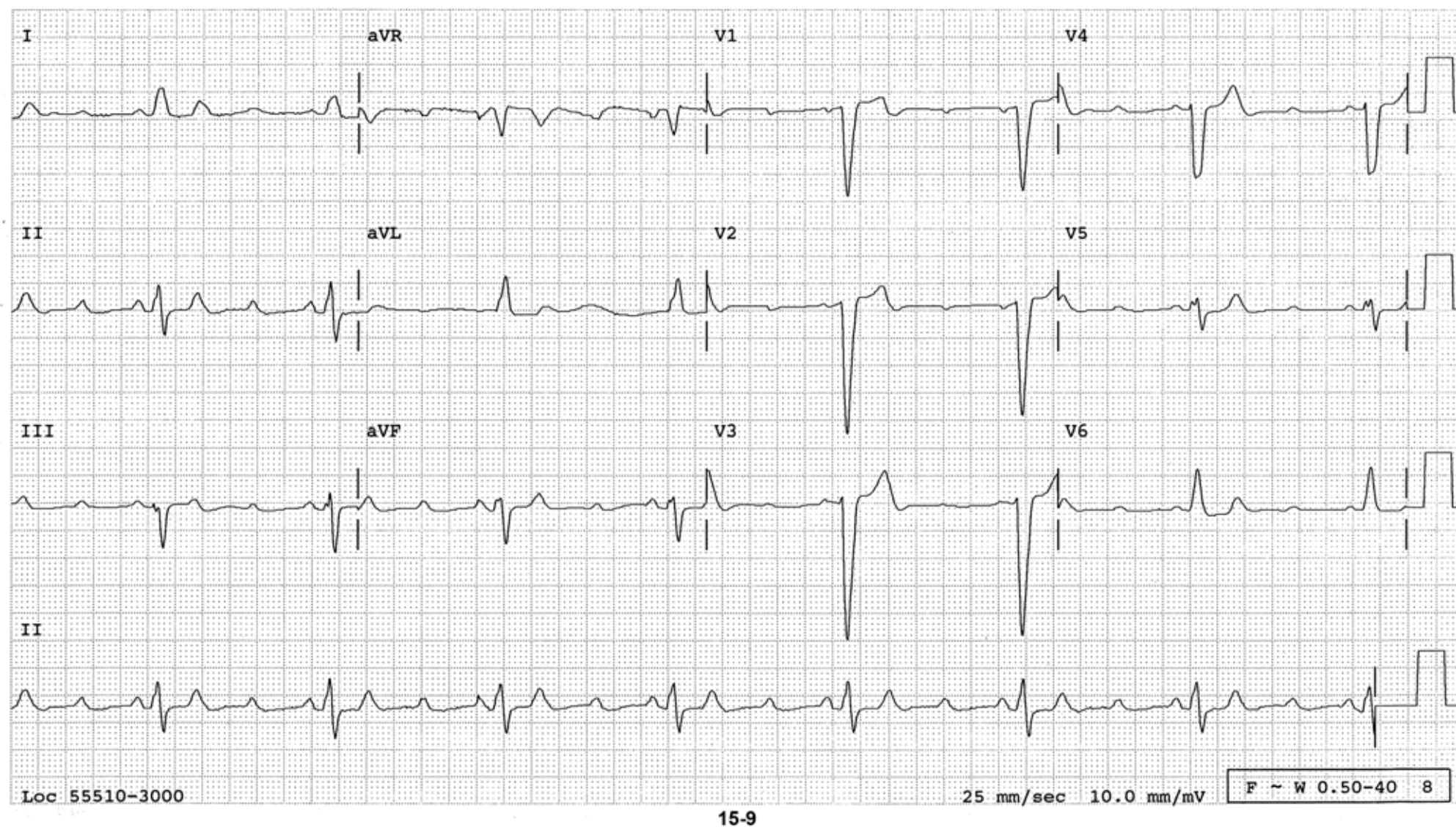
15-7 Initial NSR is followed by many P waves that are blocked. When two or more consecutive P waves are blocked with no adequate escape rhythm, the condition is called high grade AV block. In the first half of the fourth strip, the wide QRS complexes occur regularly with no fixed relationship to the P waves indicating complete AV block. However, the last four QRS complexes have a fixed PR relationship indicating 2:1 AV block. The beginning of the fifth strip is back to 1:1 conduction, then again, many P waves are blocked reflecting high grade AV block. Even when there is 1:1 conduction, the QRS is very wide indicating the presence of IVCD. Severe electrolyte imbalance or metabolic derangements can be responsible for these ECG findings. If that is the case, as the underlying abnormalities are corrected, these ECG findings should normalize as well.

- Dx:
1. High grade AV block and 3° AV block
 2. IVCD



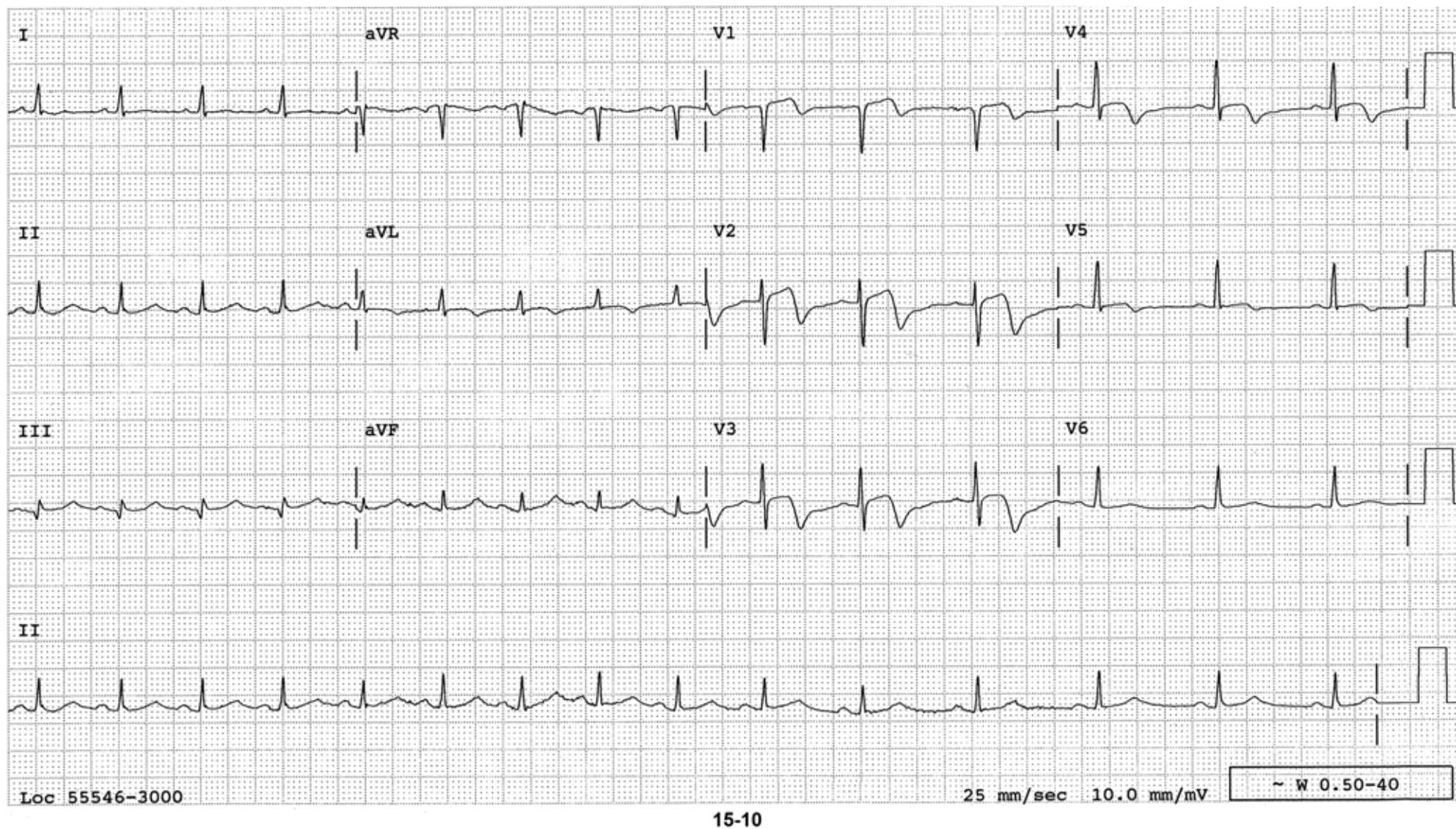
15-8 Sinus tachycardia at a rate of 116/minute. RAD, tall R wave in V_1 , ST-T changes in the right precordial leads, and deep S wave in V_6 are all diagnostic features of RVH. R waves are hardly present in V_2 - V_3 suggesting this RVH is 2° to chronic obstructive pulmonary disease (COPD). Electrical alternans in V_2 - V_3 suggests cardiac tamponade.

- Dx:*
1. Sinus tachycardia
 2. RVH, probably due to COPD
 3. Electrical alternans, consider cardiac tamponade



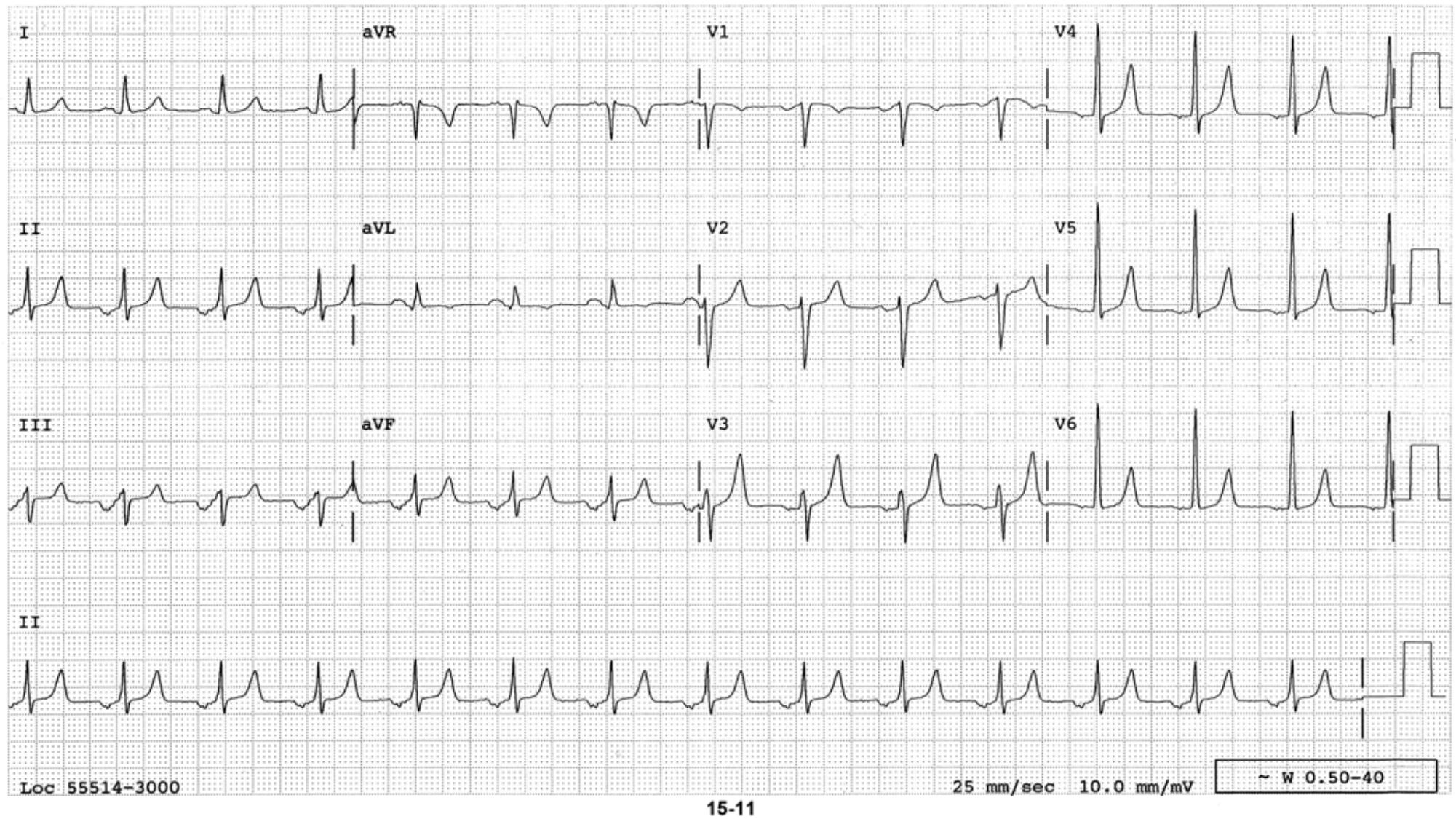
15-9 P waves occur regularly at a rate of 135/minute. Every third P wave results in a QRS with a fixed P-R interval indicating 3:1 AV block. The QRSs are wide and have typical left bundle branch pattern. The P wave is mostly negative in V_1 indicating LAE.

- Dx:
1. Sinus tachycardia with 3:1 AV block
 2. LBBB
 3. LAE



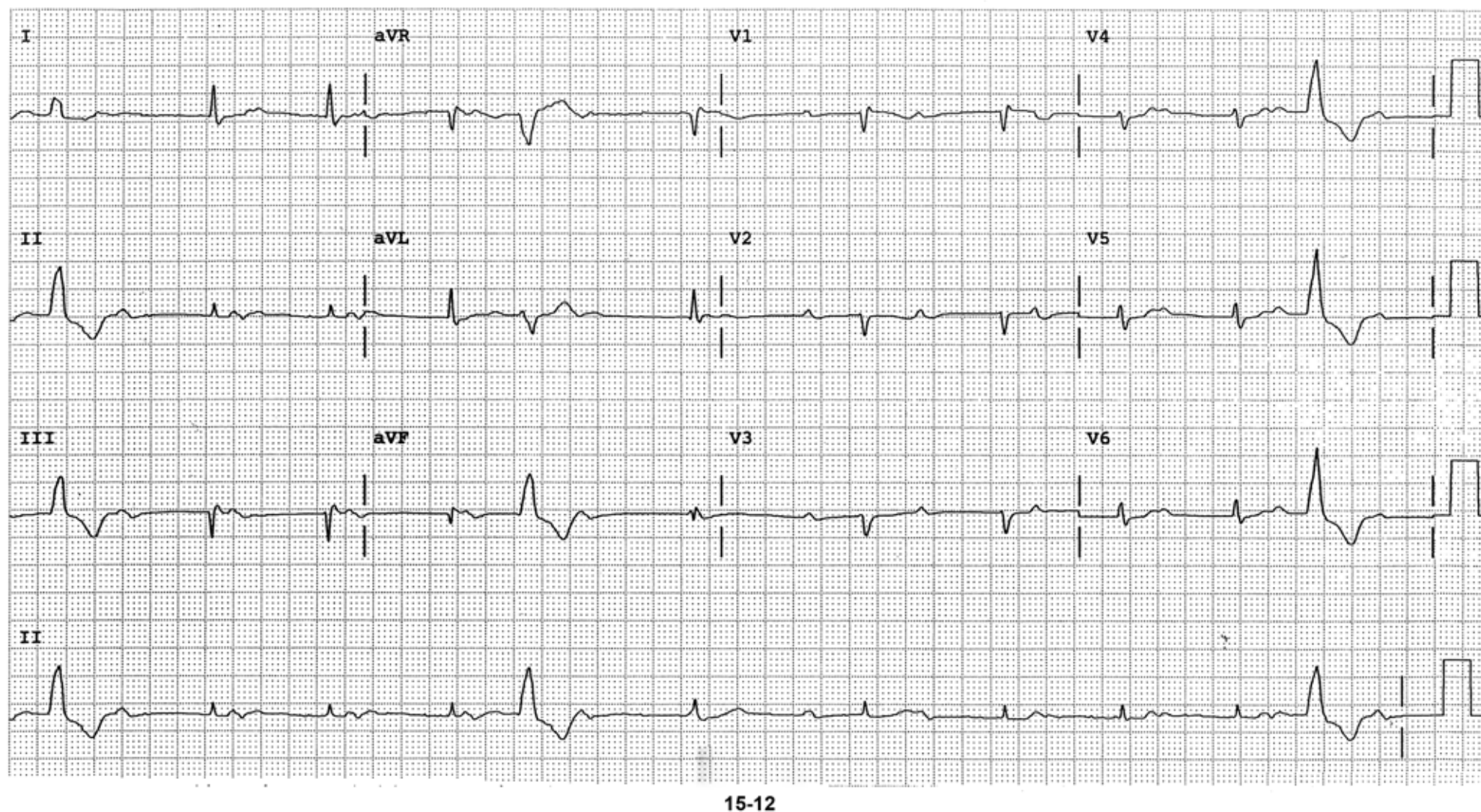
15-10 Normal sinus rhythm at a rate of 100/minute which gradually slows down to about 67/minute. Slight ST-elevation followed by T wave inversion in the precordial leads is highly suggestive of subendocardial ischemia or infarction. Q waves are present in leads III raising the possibility of old inferior MI.

- Dx:*
1. Sinus arrhythmia
 2. Probable old inferior infarct
 3. ST-T changes consistent with subendocardial ischemia or infarction of the anterior wall



15-11 A regular rhythm with narrow QRSs at a rate of 84/minute. P waves are entirely negative in the inferior leads indicating that the atria are depolarized retrograde. Since the P-R interval is not short and measures about 150 milliseconds, low atrial rhythm will be considered rather than AV junctional rhythm. Low atrial rhythm is benign. The base of the tall T waves become quickly narrow, making the T wave tented, narrow and pointed, suggesting hyperkalemia.

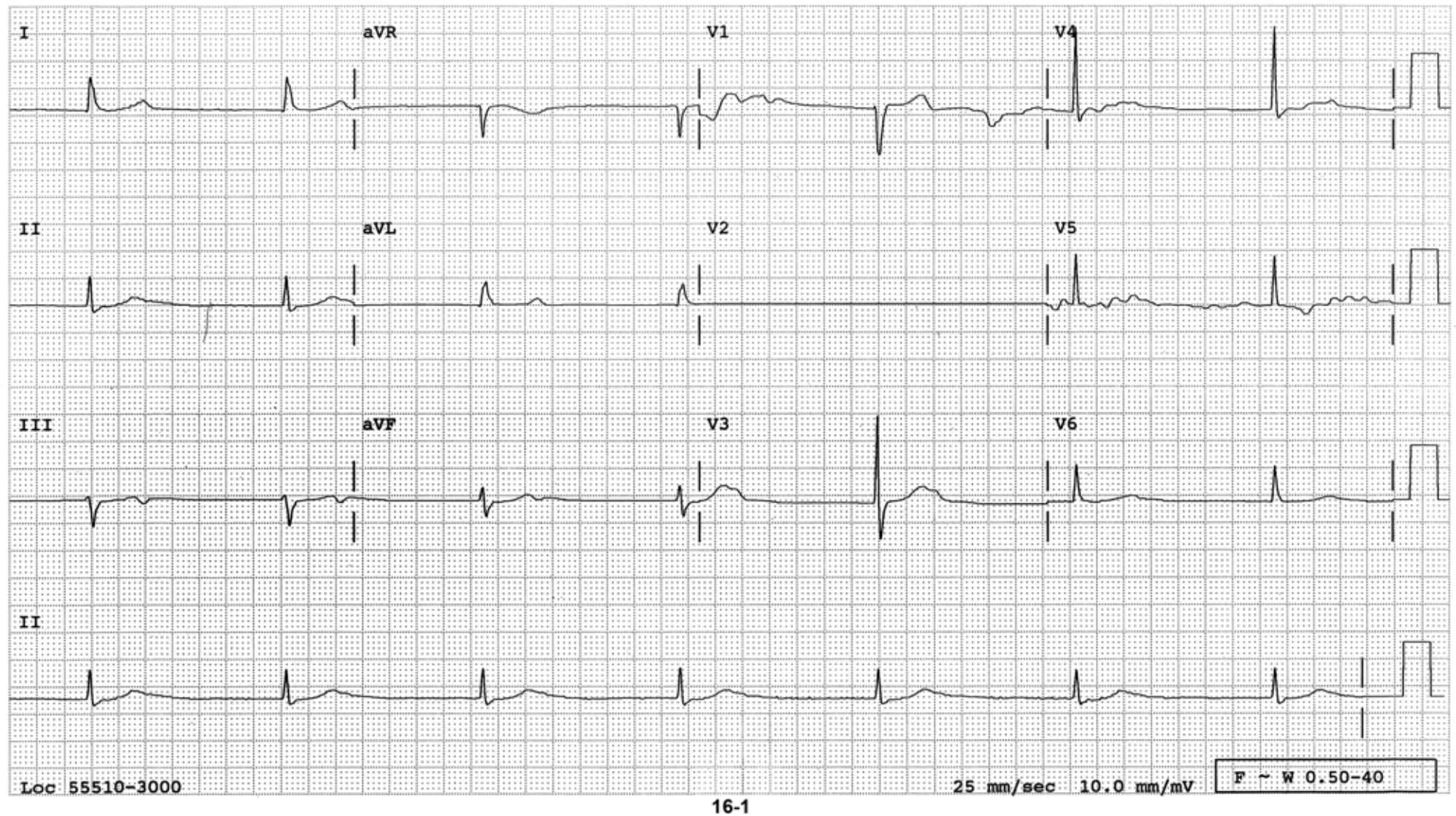
- Dx:
1. Low atrial rhythm, benign
 2. Consider hyperkalemia



15-12 A narrow QRS rhythm is interrupted by frequent PVCs. Regularly occurring P waves are easily recognizable in the right precordial leads as well as in the rhythm strip of lead II. Progressive lengthening P-R interval can be appreciated indicating AV Wenckebach phenomenon. It is not a complete AV block since the R-R interval is not regular. In fact, the R to R interval shortens which is another feature of AV Wenckebach phenomenon. The sixth complex (the last complex of aVR, aVL and aVF) may be conducted from the preceding P wave or it may be an AV junctional escape complex. These cannot be settled from the tracing given. If it is conducted from the preceding P wave, the P-R interval measures about 760 milliseconds which is not unheard of (the world record for the longest conducted P-R interval is 1 second). When one marches the P-P interval, there is a P wave hidden within that QRS which is the blocked P wave of this Wenckebach cycle. The Q wave in lead III is deep and wide enough indicating old inferior infarct. Poor progression of the R waves in the precordial leads suggests old AMI as well.

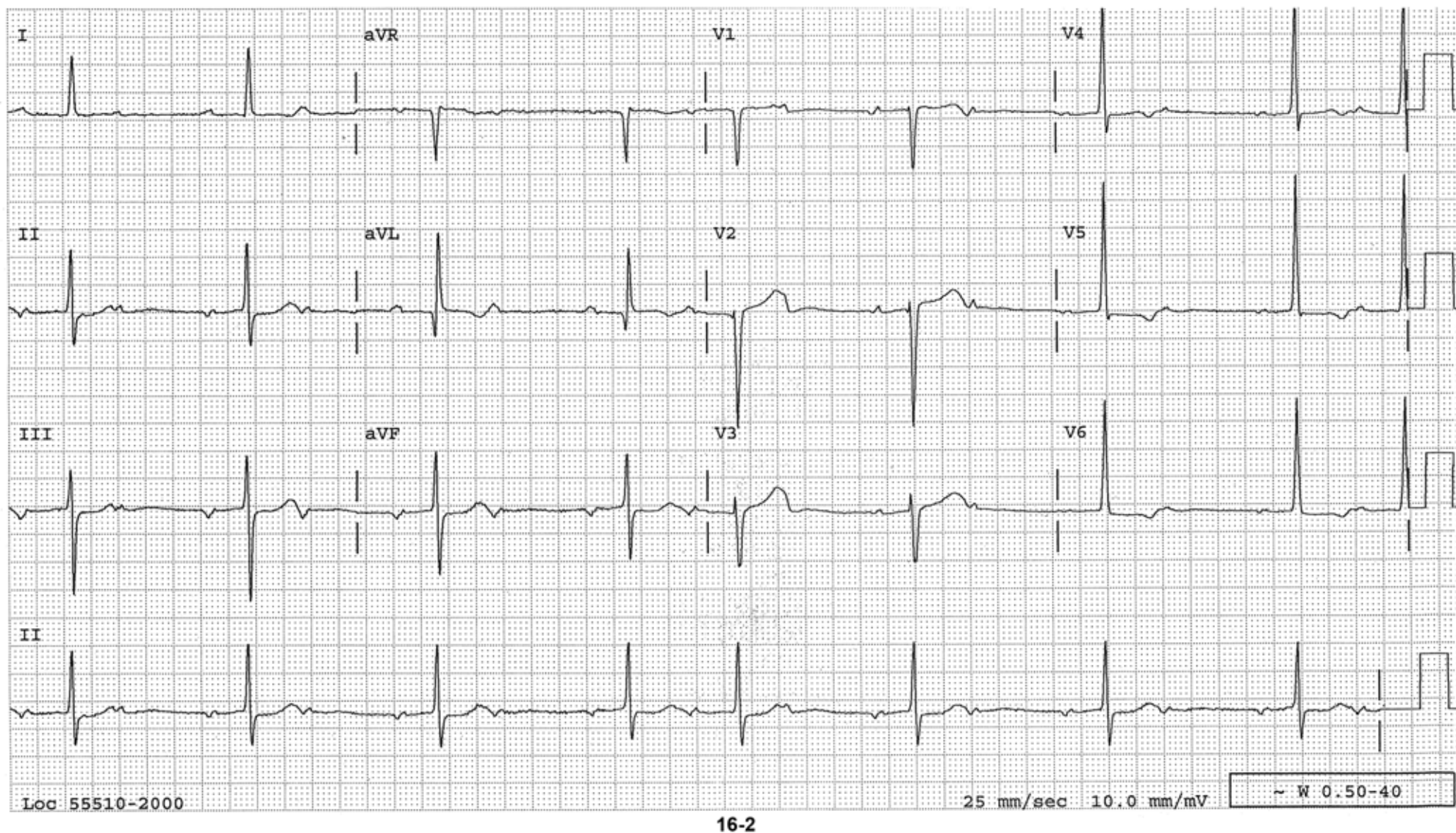
- Dx:
1. Sinus rhythm with Type I 2° AV block
 2. Frequent PVCs
 3. Old inferior infarct
 4. Consider old anteroseptal infarct

SECTION 16



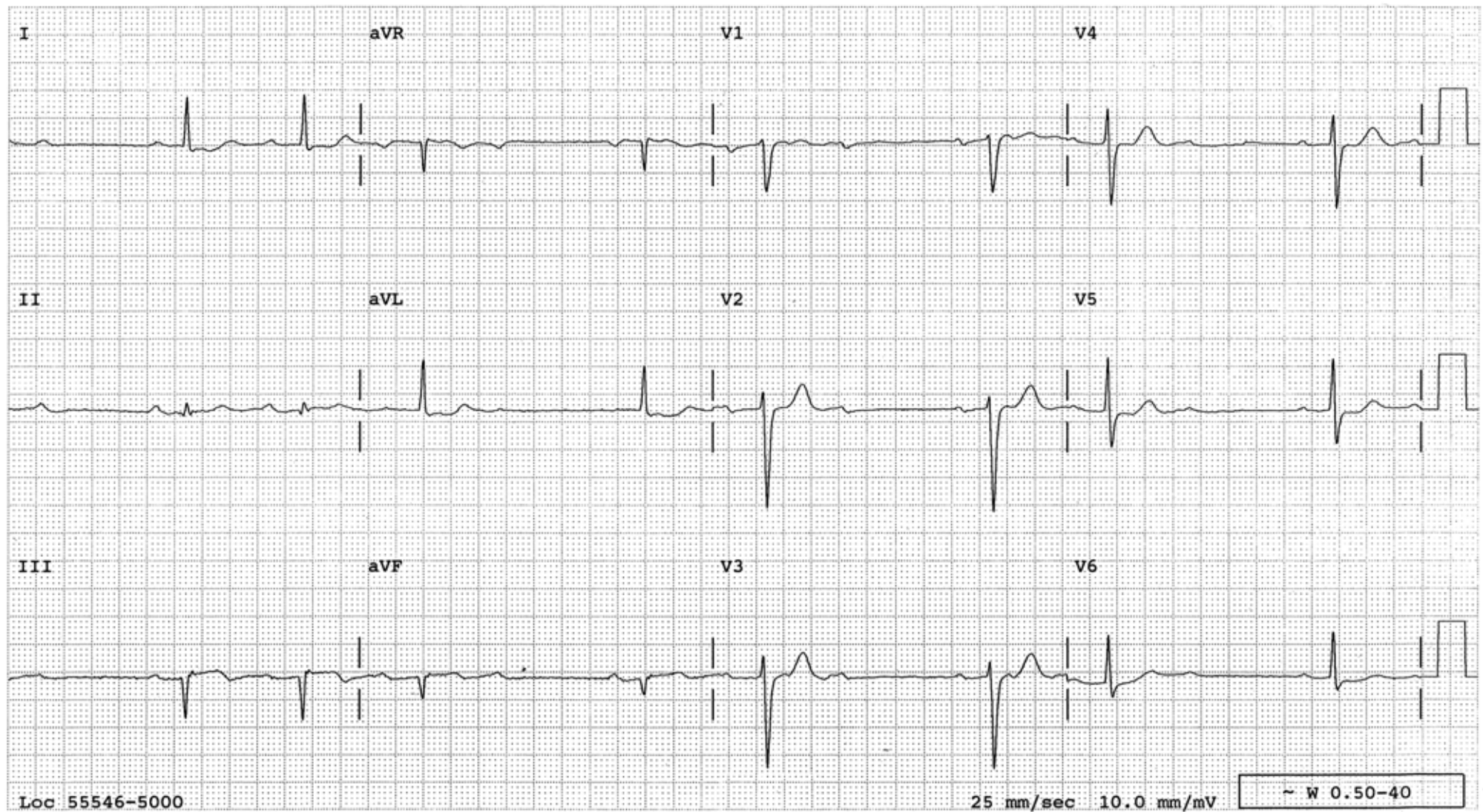
16-1 A regular rhythm at a rate of 42/minute. The QRSs are narrow and no distinctive P waves can be identified. The terminal negativity in lead II is most likely a negative P wave. The bump on the downstroke of the T wave in V₃ is more likely a part of the T wave rather than an anterograde P wave. The primary disorder in this patient is sinus slowing which allows the AV junctional pacemaker to escape.

Dx: Sinus node dysfunction and AV junctional escape rhythm



16-2 2:1 AV block is present initially, then in the middle of the tracing, 3:2 AV Wenckebach phenomenon, then back to 2:1 AV block. Here, 2:1 conduction ratio can be considered as the shortest Wenckebach cycle, i.e. the conduction ratio of 5:4 becomes 4:3, then 3:2, and finally 2:1. Thus, during 2:1 AV block, if a typical Wenckebach phenomenon is manifested somewhere in the tracing, it is Type I all along, not Type II, then Type I, then back to Type II. If Wenckebach phenomenon is not seen during 2:1 AV block, pay attention to the QRS width and the P-R interval of conducted beats; if the QRS is narrow and the P-R interval is long, the block is most likely within the AV node. Otherwise, the block is below the His bundle. This distinction is important because the block within the AV node is mostly due to reversible conditions and relatively benign, while the block below the His bundle is not due to reversible conditions and more troublesome, often requiring a pacemaker. Findings for LVH are present.

- Dx: 1. Type I 2° AV block all along
2. LVH



16-3

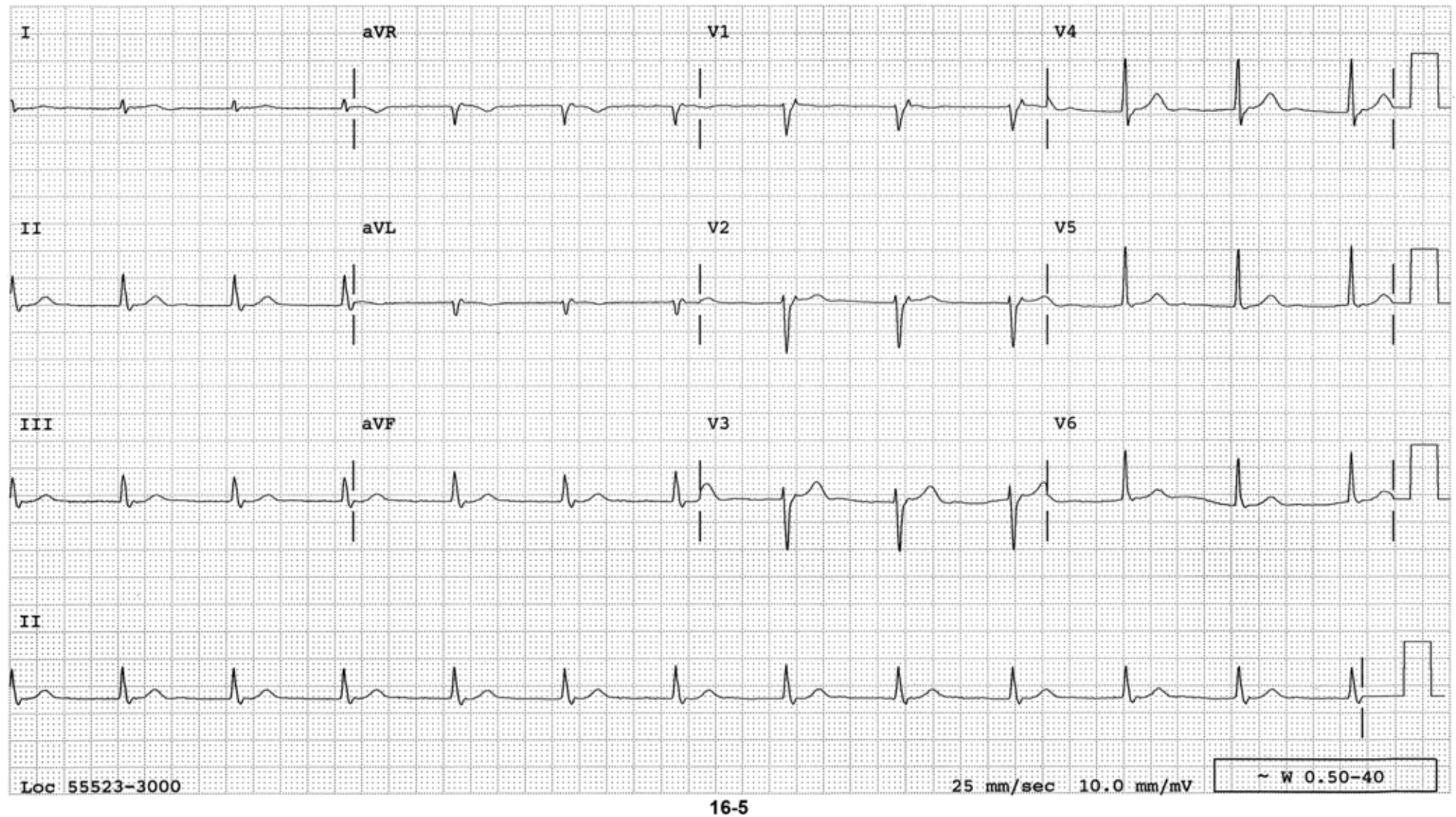
16-3 P waves occur regularly at a rate of 56/minute. The P-R interval progressively lengthens and either the fourth or the third P wave is blocked indicating Type I 2° AV block. Q waves, ST elevation and T wave inversion in lead III indicate inferior STEMI, which is the cause of this AV block. 2° AV block in inferior infarction is Type I and is almost always transient, while that in anterior MI is Type II and is almost always permanent. When 2° AV block progresses to 3° AV block, the escape rhythm is AV junctional in inferior MI and ventricular in anteroseptal MI.

- Dx:
1. Sinus rhythm with Type I 2° AV block
 2. Inferior infarct as the cause of this AV block



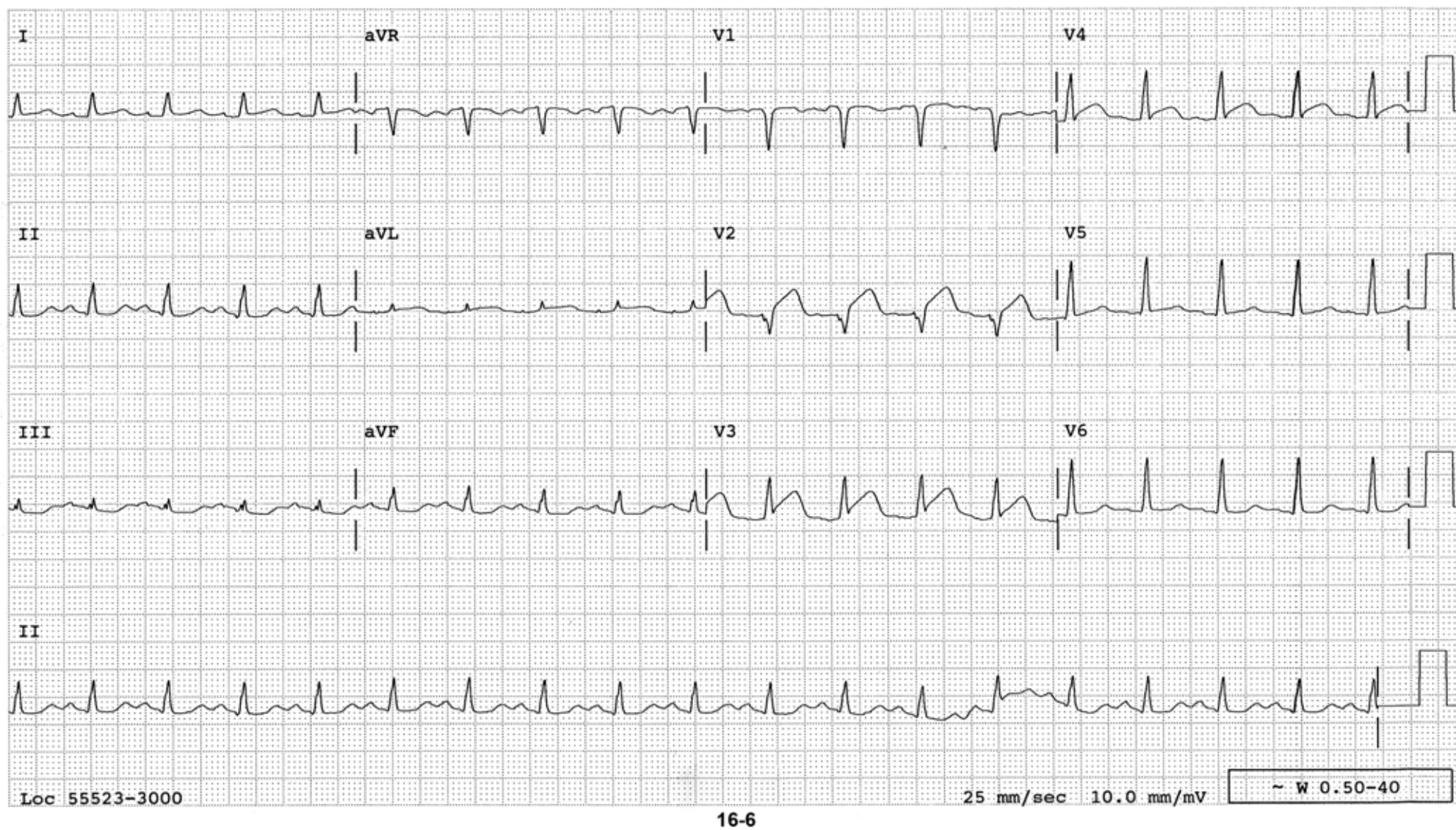
16-4 A regular sinus rhythm is present. Occasionally the P waves are not conducted to the ventricle without any progressive lengthening P-R interval prior to the block indicating Type II 2° AV block.

Dx: Sinus rhythm with Type II 2° AV block



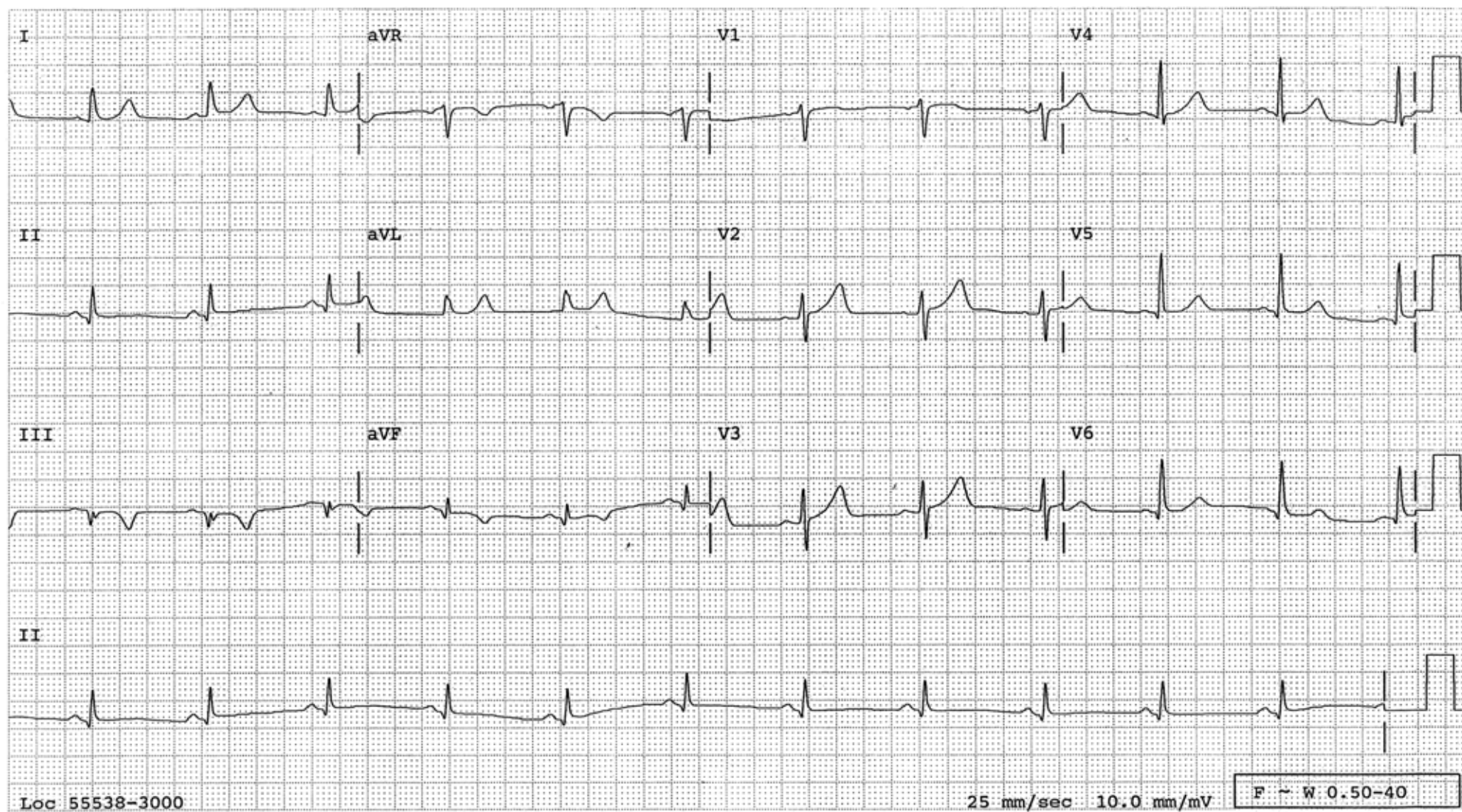
- 16-5 A regular rhythm at a rate of 73/minute. The QRSs are narrow. There are no P waves in front of the QRS in any leads. The terminal negative deflection in the inferior leads and a positive blip at the end of the QRS in V_1 and V_2 most likely reflect retrograde P wave from AV junctional rhythm (pseudo S wave in the inferior leads and pseudo R' in V_1 and V_2). The rate of 73 is faster than the intrinsic AV junctional rate of 40-50, but not fast enough (> 130/minute) to be called junctional tachycardia and this rhythm will be called accelerated junctional rhythm.

Dx: Accelerated junctional rhythm with retrograde 1:1 conduction to the atria



16-6 Sinus tachycardia at a rate of 110/minute. Coved ST elevation with Q waves in the right precordial leads indicates acute AMI.

- Dx: 1. Sinus tachycardia
2. Acute antero-septal infarct

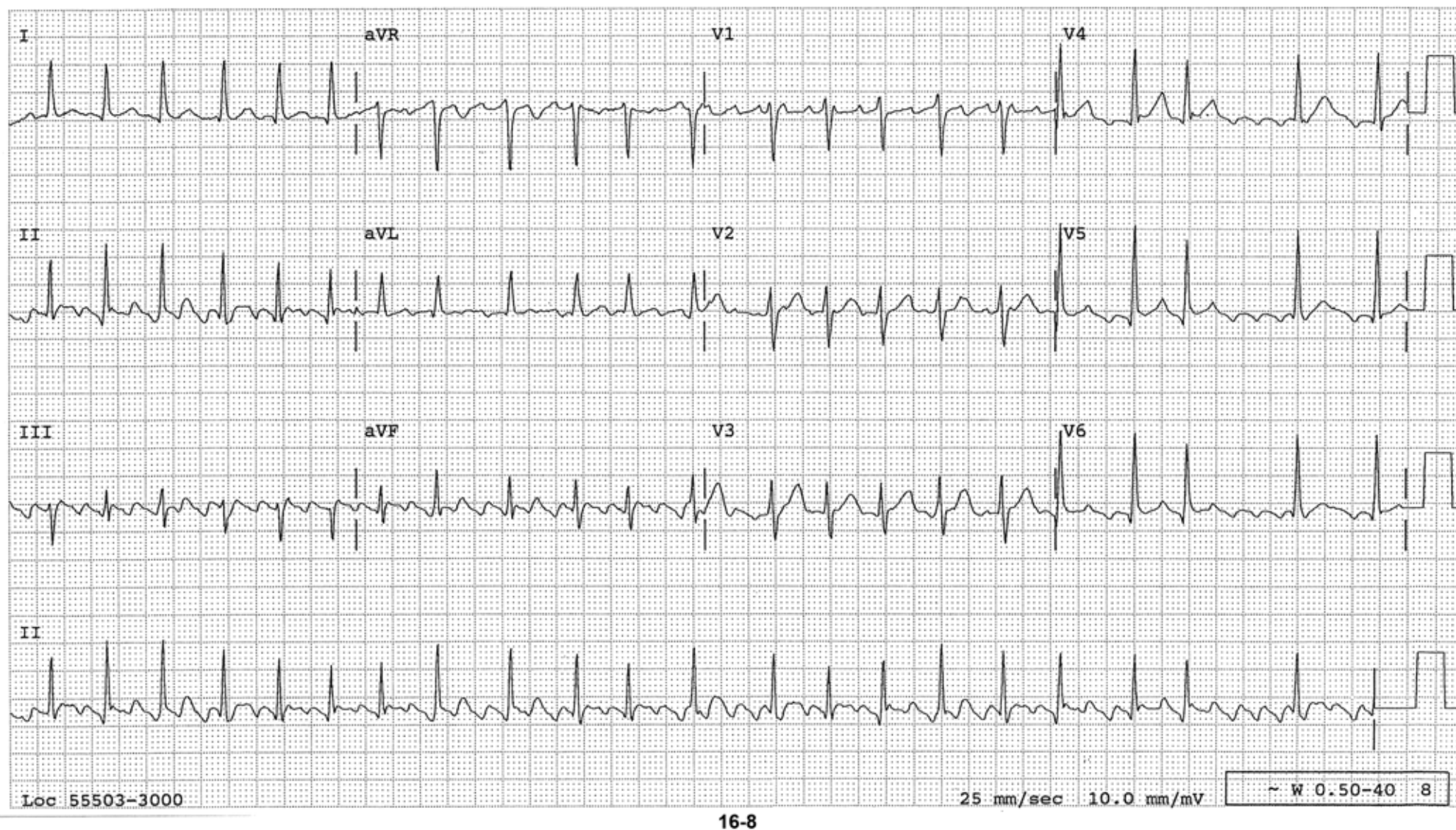


16-7

16-7 Normal sinus rhythm at a rate of 70/minute. Q waves with T wave inversion in the inferior leads indicate inferior MI, probably recent. No other abnormalities are noted.

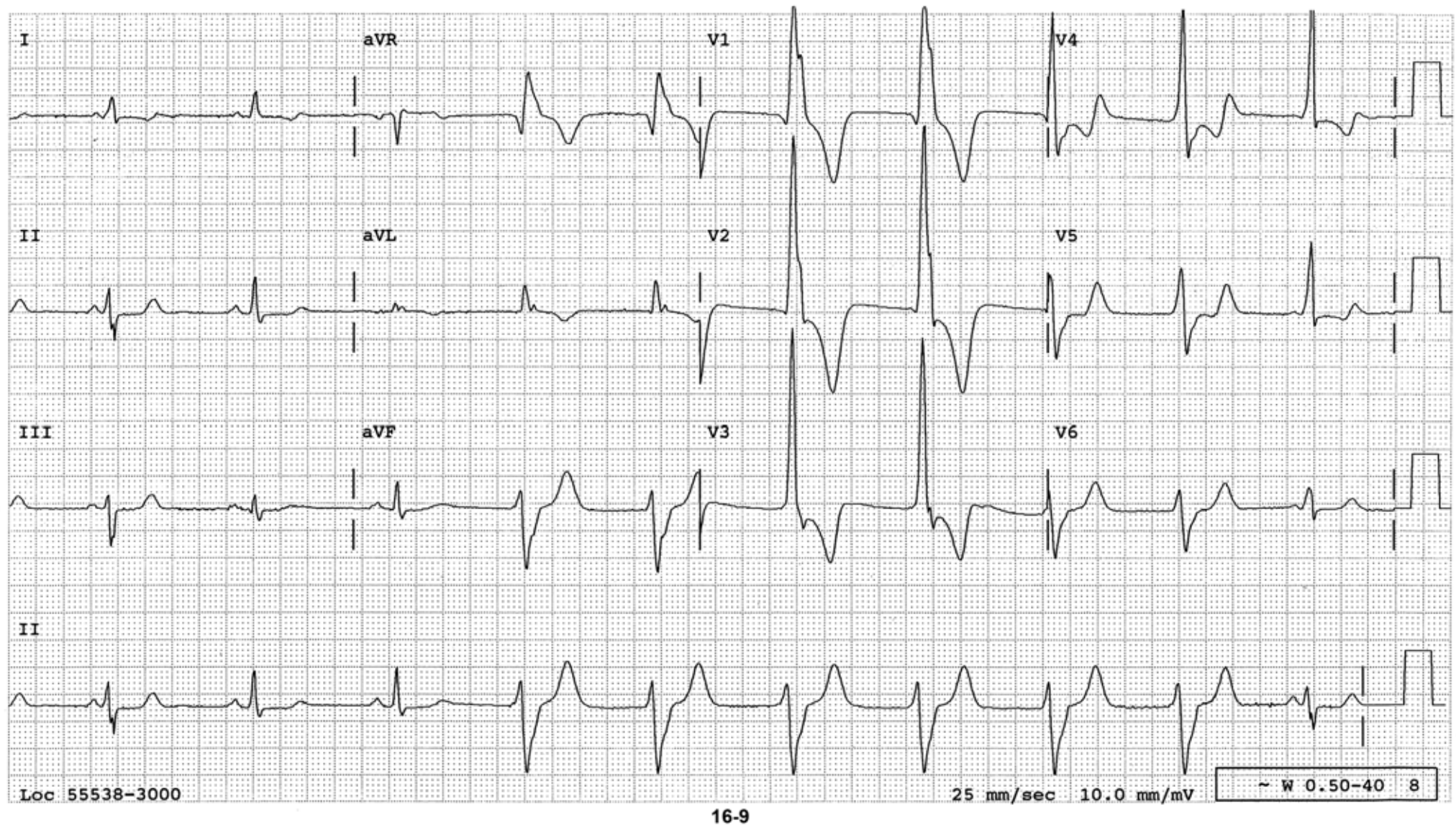
Dx: 1. NSR

2. Inferior infarct, probably recent



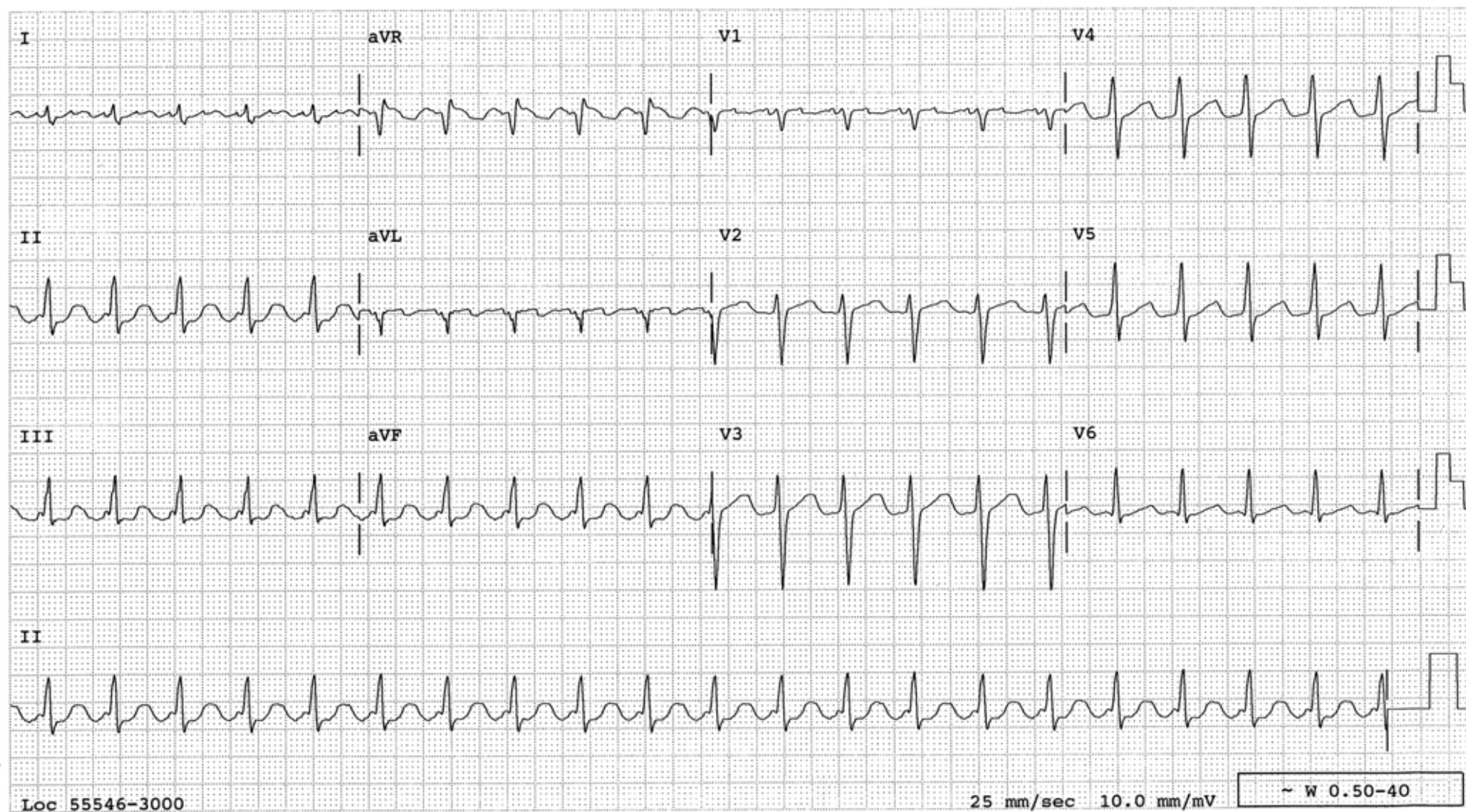
16-8 Irregularly irregular rhythm at ~140/minute suggestive of atrial fibrillation. However, there are definite flutter waves, more convincingly near the end of the tracing. Atrial fibrillation may have coarse fibrillatory waves but not so well-organized up-down, up-down atrial activities like this and this is an example of atrial flutter with varying AV conduction ratio. Thus, QRSs in atrial flutter can occur irregularly irregular at times. Paying attention to the baseline during long R-R intervals can be useful.

Dx: Atrial flutter with varying AV conduction ratio resulting in an irregularly irregular rhythm



16-9 Initially, NSR is present which is followed by a regular wide QRS rhythm at a rate of 60/minute with no P waves; hence a ventricular rhythm. P waves are buried within the QRSs during this period until finally it emerges in front of the QRS. The first and last QRS complexes are different from the normally conducted sinus complexes (second and third QRSs) or from the complexes during the wide QRS rhythm indicating that these are fusion complexes; fusion of sinus and ventricular complexes. Since the rate of this ventricular rhythm is faster than the intrinsic ventricular rate, but not fast enough (> 130/minute) to be called VT, this will be called accelerated idioventricular rhythm. This rhythm is primarily seen during acute myocardial ischemia or infarction, and often it is a sign of reperfusion.

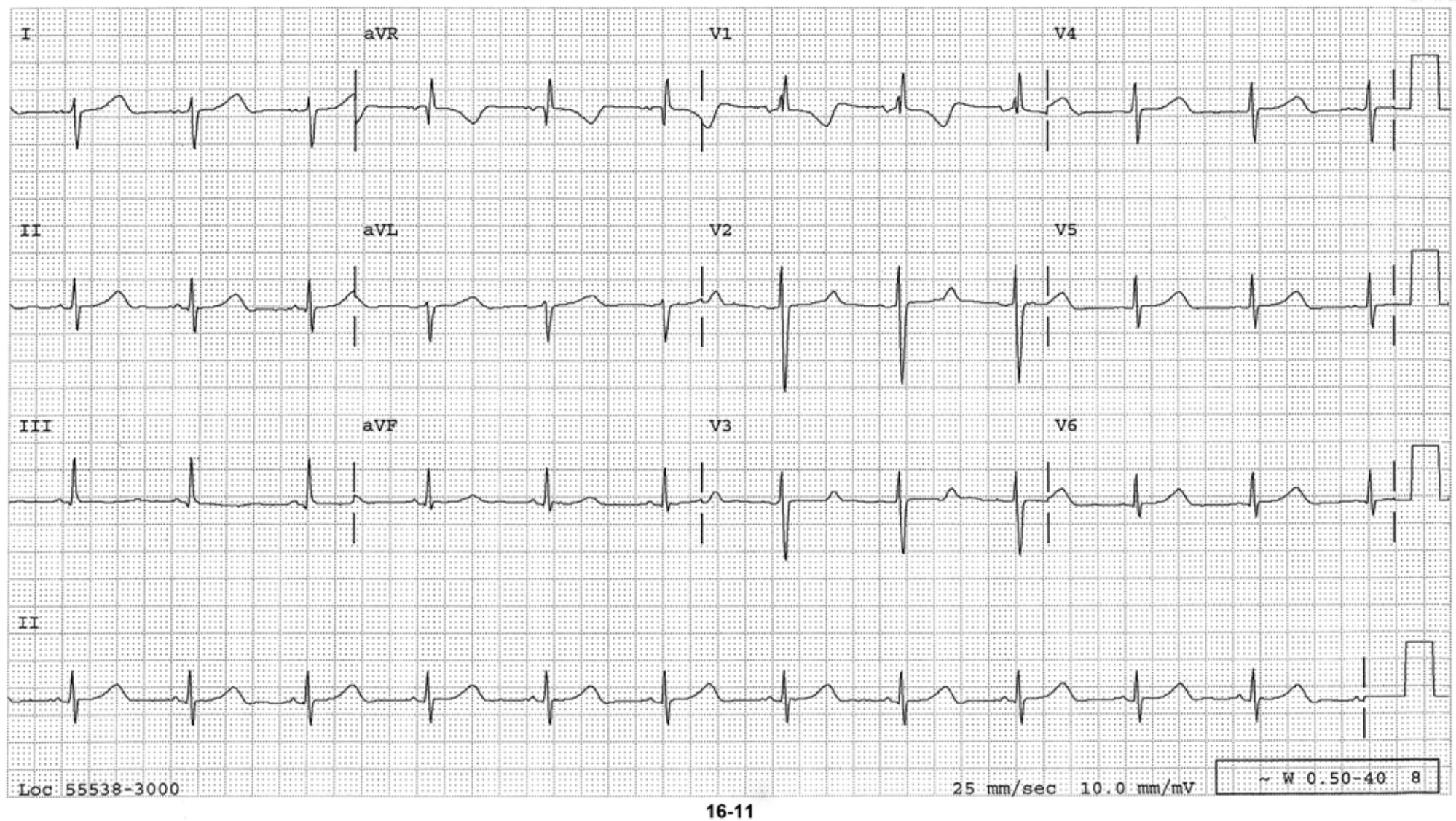
- Dx:*
1. A short run of accelerated idioventricular rhythm between NSR
 2. Two fusion complexes



16-10

16-10 A narrow QRS tachycardia at a rate of 125/minute. Two atrial activities are clearly visible between the QRSs in V_1 at a rate of 250/minute. This will force us to look for the flutter waves in the inferior leads which are indeed there as dome, dome, dome, dome.

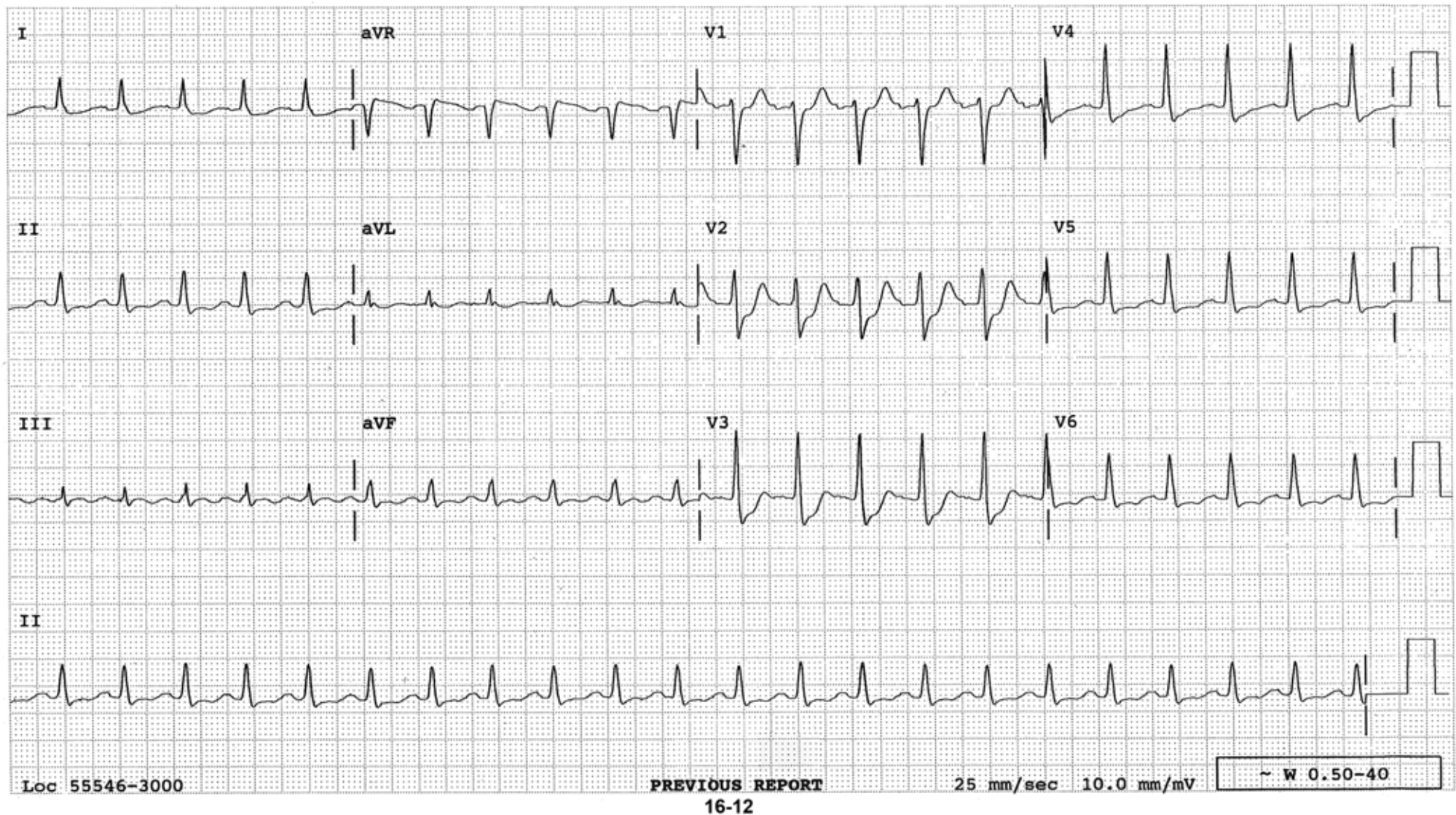
Dx: Atrial flutter with 2:1 AV conduction



16-11 Normal sinus rhythm at 69/minute. Narrow QRS (80 milliseconds) with rsR' in V₁ reflecting incomplete RBBB. That combined with RAD is how atrial septal defect (ASD) secundum manifests. If it is ASD primum, the QRS axis will show left axis deviation.

Dx: 1. NSR

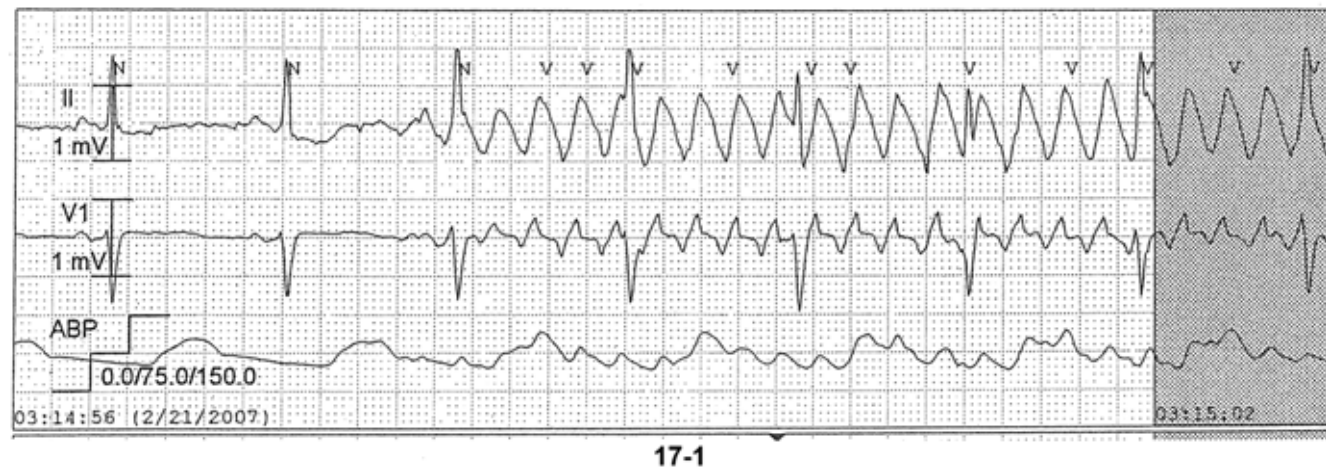
2. Consider ASD, secundum



16-12 Sinus tachycardia at a rate of 135/minute. Somewhat tall R waves and horizontal ST depression in V_2 and V_3 are reciprocal changes of "injury pattern" ST elevation of the posterior wall. This is best appreciated by looking at the tracing from the back while holding it upside down against the light. More often, a posterior infarct occurs as a part of inferoposterior or posterolateral MI, in which case, the inferior leads or lateral leads would reveal the infarct pattern as well. At times, the infarct may involve only the posterior wall as in this case.

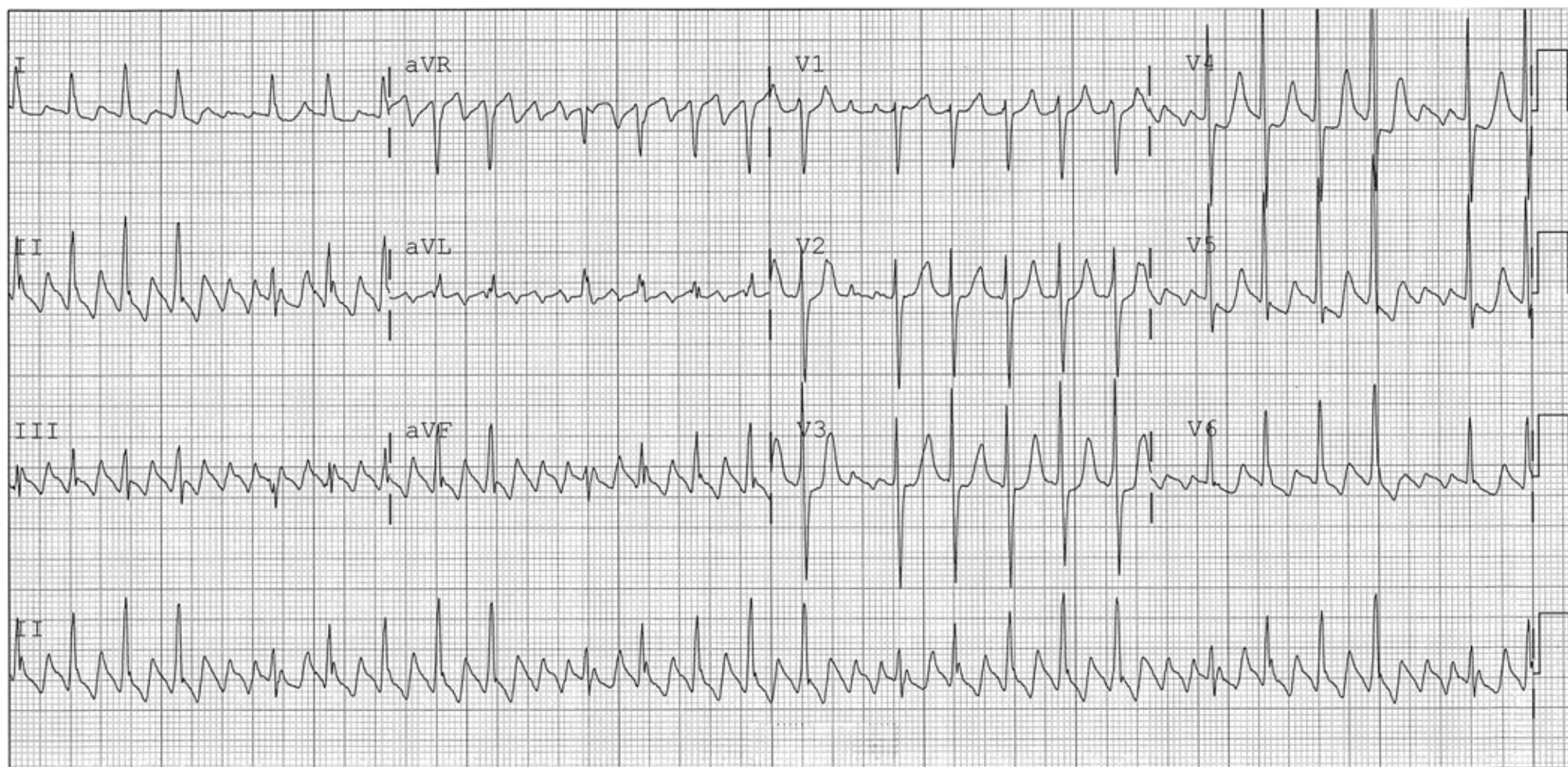
Dx: Acute posterior STEMI

SECTION 17



- 17-1 This tracing suggests either atrial flutter or VT. It is not atrial flutter, however. If it were, the QRS complexes occurring regularly would mean there is a fixed AV conduction ratio. If so, the QRS complexes and the flutter waves would maintain a fixed relationship. That is not happening here. It is not VT because the narrow QRS complexes march through. This tracing is an example of muscle tremors simulating either atrial flutter or VT.

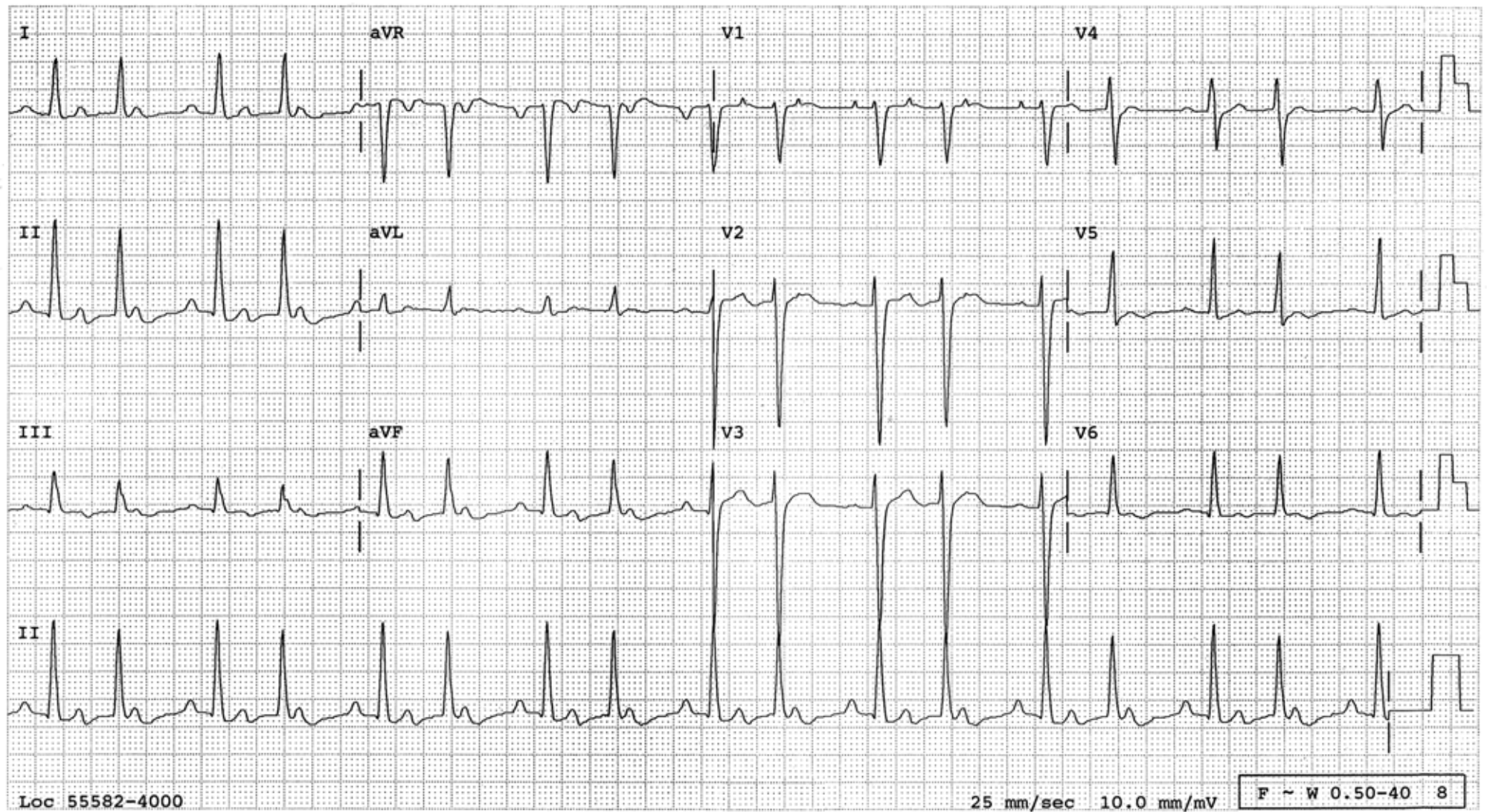
Dx: Artifact simulating either atrial flutter or VT during a regular sinus rhythm



17-2

17-2 The sawtooth pattern of atrial flutter is obvious in the inferior leads. 2:1 AV conduction becomes 4:1 after five beats, resulting in five beats being grouped together. During the cycle of five beats, each QRS complex in the sequence maintains fixed relationship with the flutter wave, confirming that these are indeed flutter waves, not artifact. The reason why the QRS complex height changes is because it is summated with different height of the flutter wave.

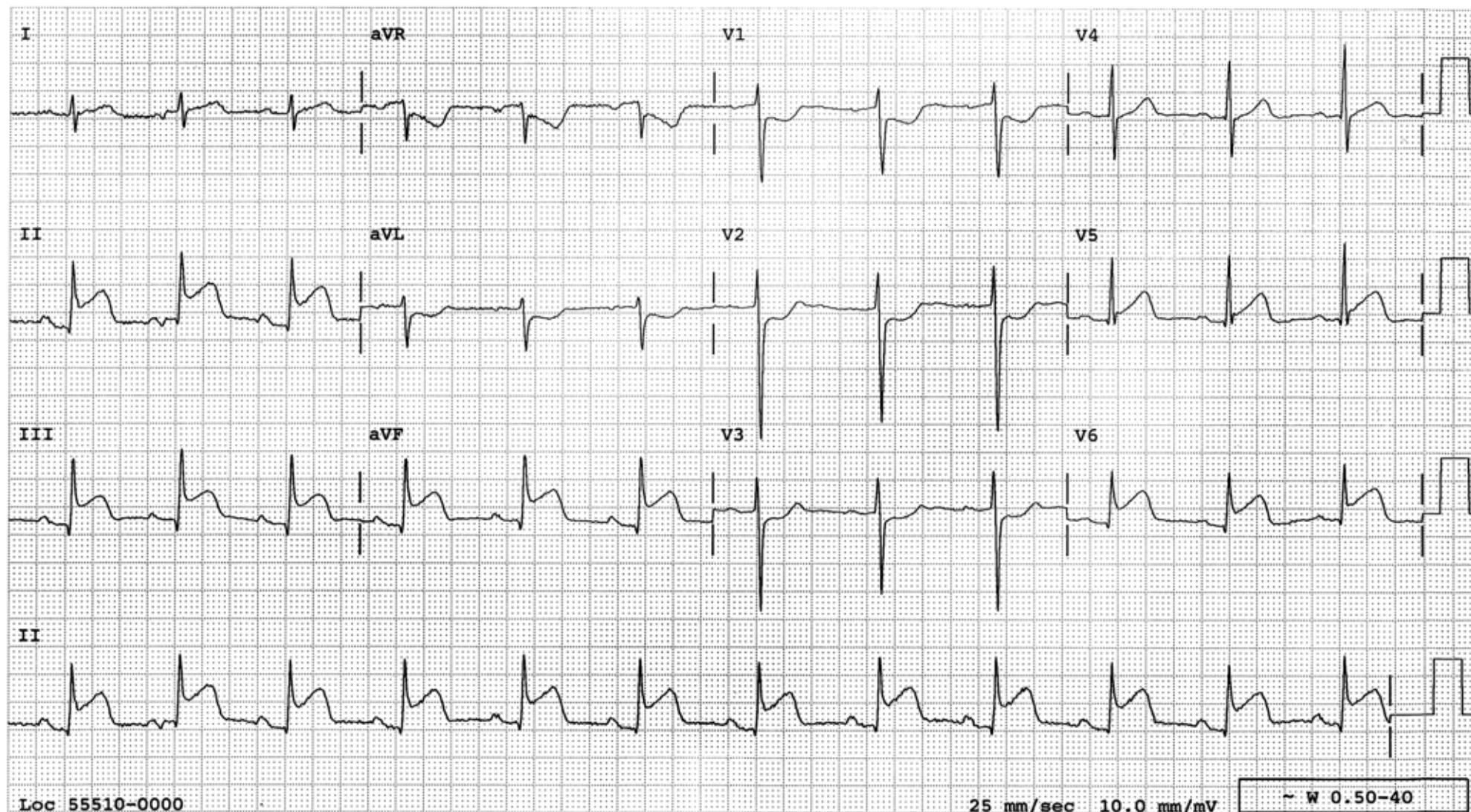
Dx: Atrial flutter



17-3

17-3 A regular atrial rhythm at a rate of 145/minute. The P wave is small, narrow and entirely positive in V₁ which is different from ordinary P waves during sinus rhythm, and most likely this is an ectopic atrial tachycardia. There is a 3:2 AV Wenckebach phenomenon. A 3:2 AV Wenckebach phenomenon will result in the QRSs being paired as in this case. Atrial tachycardia with a variable AV conduction is often caused by digitalis toxicity. Voltage criteria and ST-T changes for LVH are present.

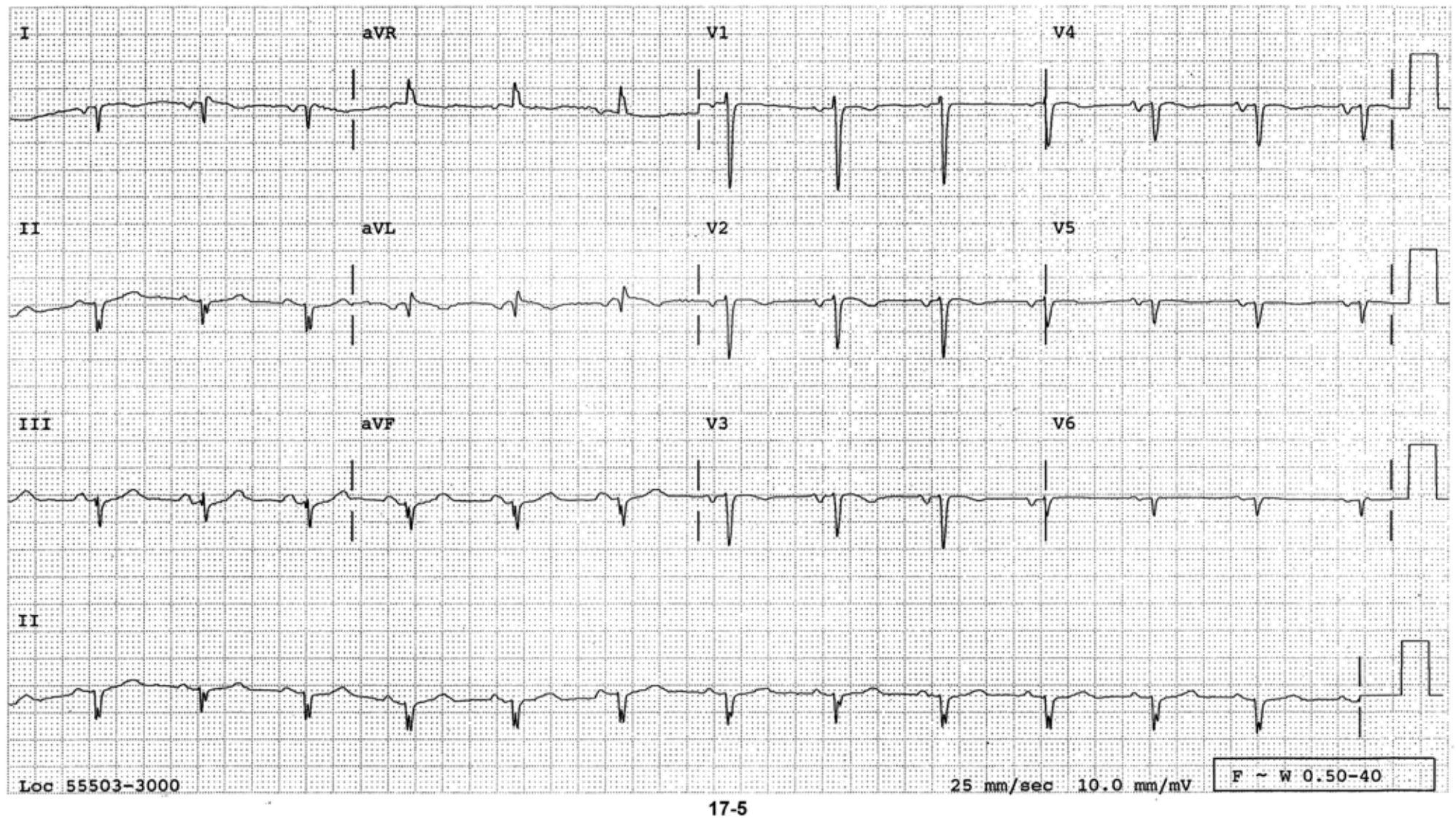
- Dx: 1. Atrial tachycardia with a 3:2 AV Wenckebach phenomenon. Consider digitalis toxicity if the patient is taking digitalis
2. LVH



17-4

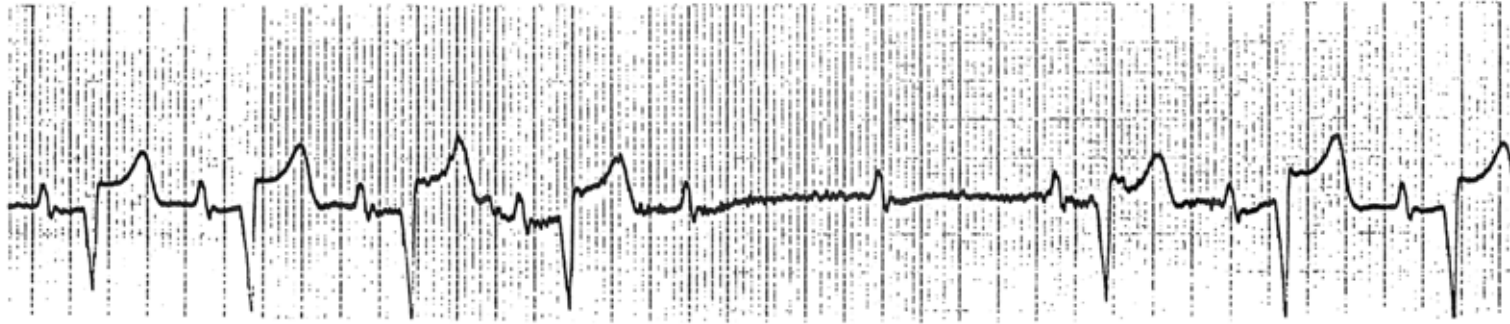
17-4 Normal sinus rhythm at 72/minute ST elevation in inferior leads and V_4 - V_5 combined with horizontal ST depression in V_1 - V_3 is diagnostic of acute inferoposterolateral MI. ST-segment is reciprocally depressed only in aVL, not in lead I, indicating the ST vector is pointed more or less straight down, not down and to the right, indicating RV is not involved. Therefore the culprit lesion is not in proximal RCA but either in the circumflex coronary artery or RCA not proximal.

- Dx: 1. NSR
2. Acute inferoposterolateral MI



17-5 Normal sinus rhythm at a rate of 78/minute. The P wave, QRS and T wave are all inverted in lead I. Only two things will do this: reversed arm leads or dextrocardia. The findings in the precordial leads will settle the issue. If the R waves progress normally, then it is the former. If the R waves regress as in this case and the P waves remain either negative or biphasic in the left precordial leads, it is the latter.

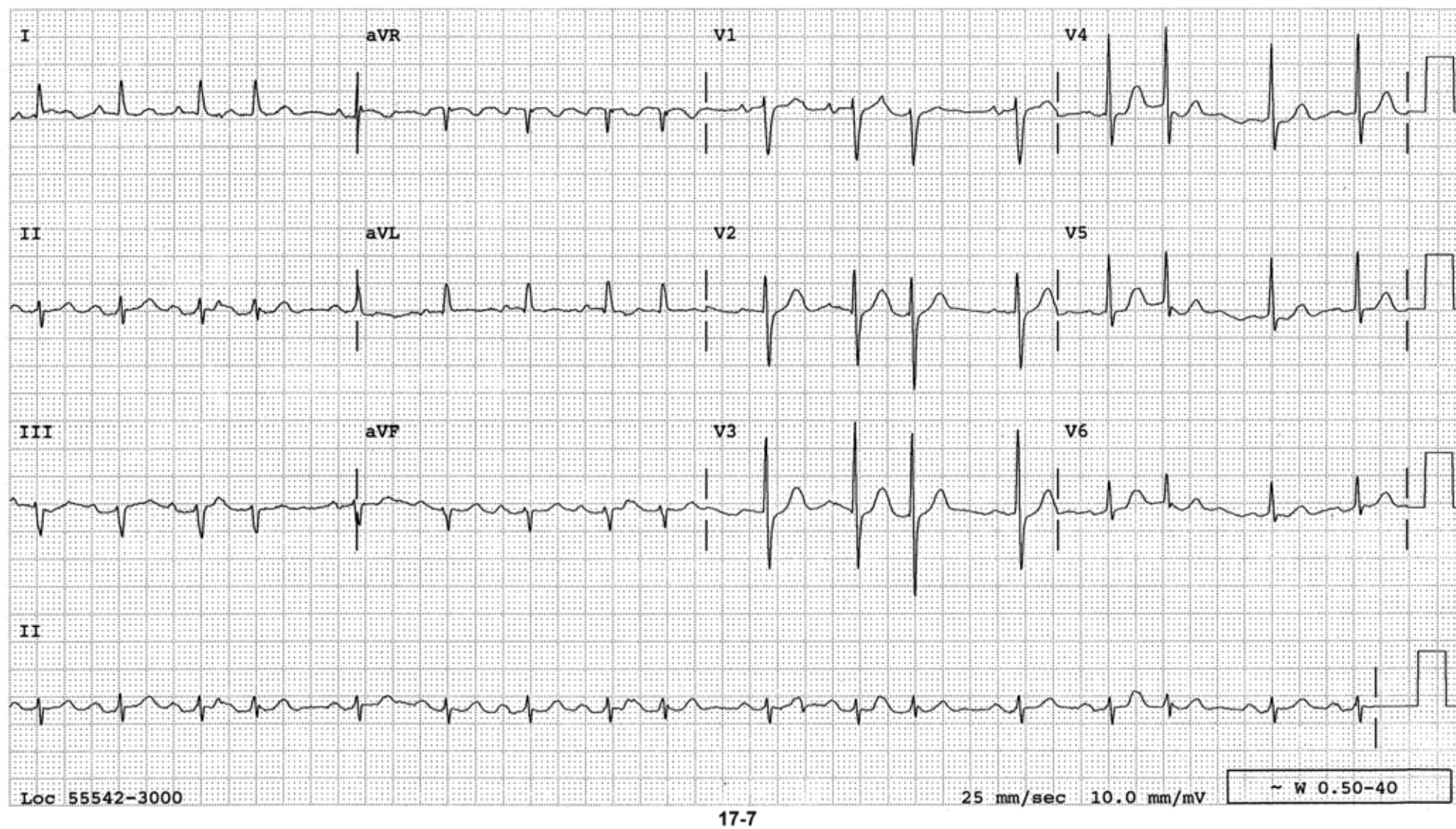
- Dx: 1. NSR
2. Dextrocardia



17-6

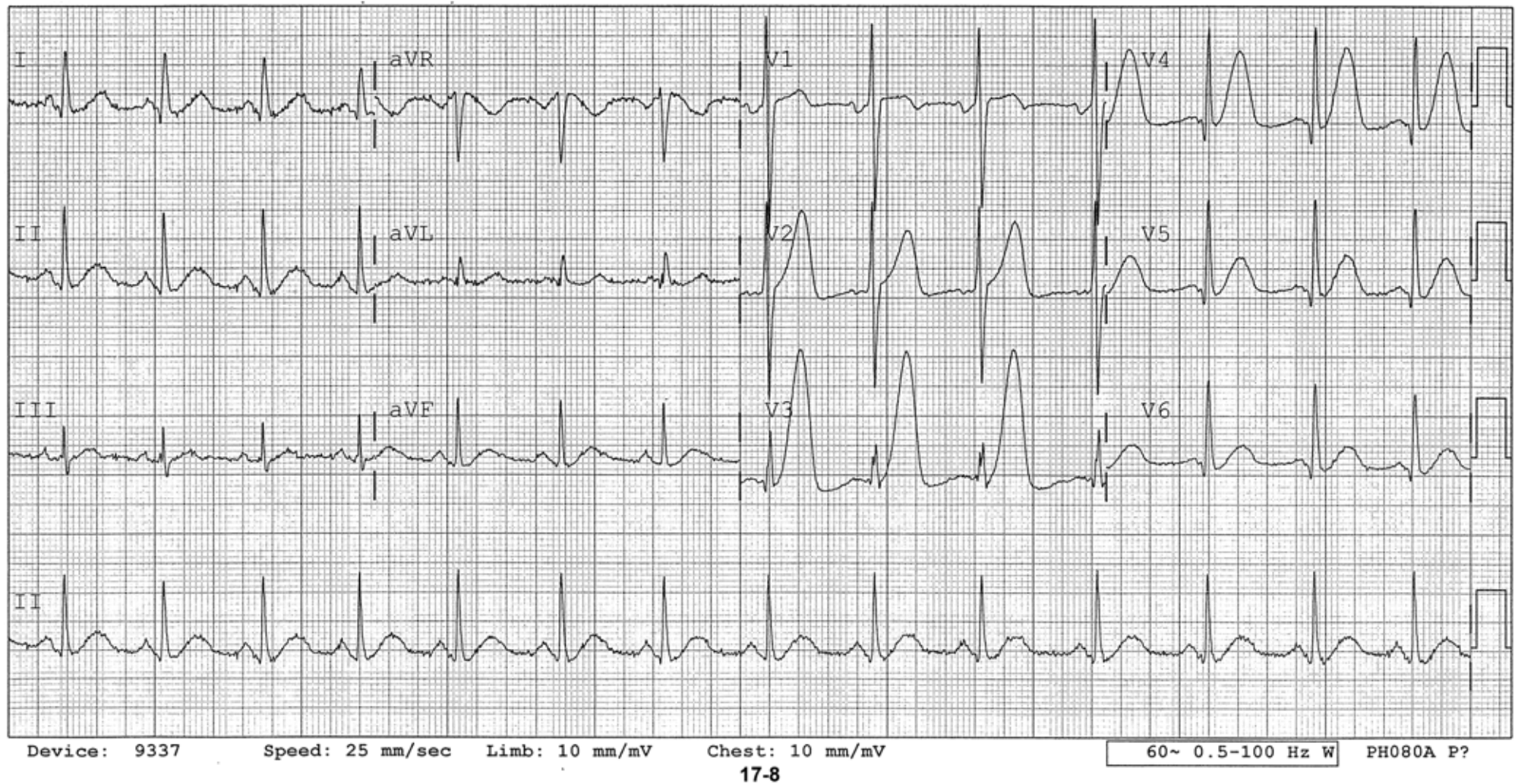
17-6 Normal sinus rhythm. Two P waves are not conducted consecutively indicating high grade AV block. The P-R intervals stay the same and the QRS is somewhat wide, and the location of the block is most likely below the HIS bundle.

Dx: Type II 2° AV block



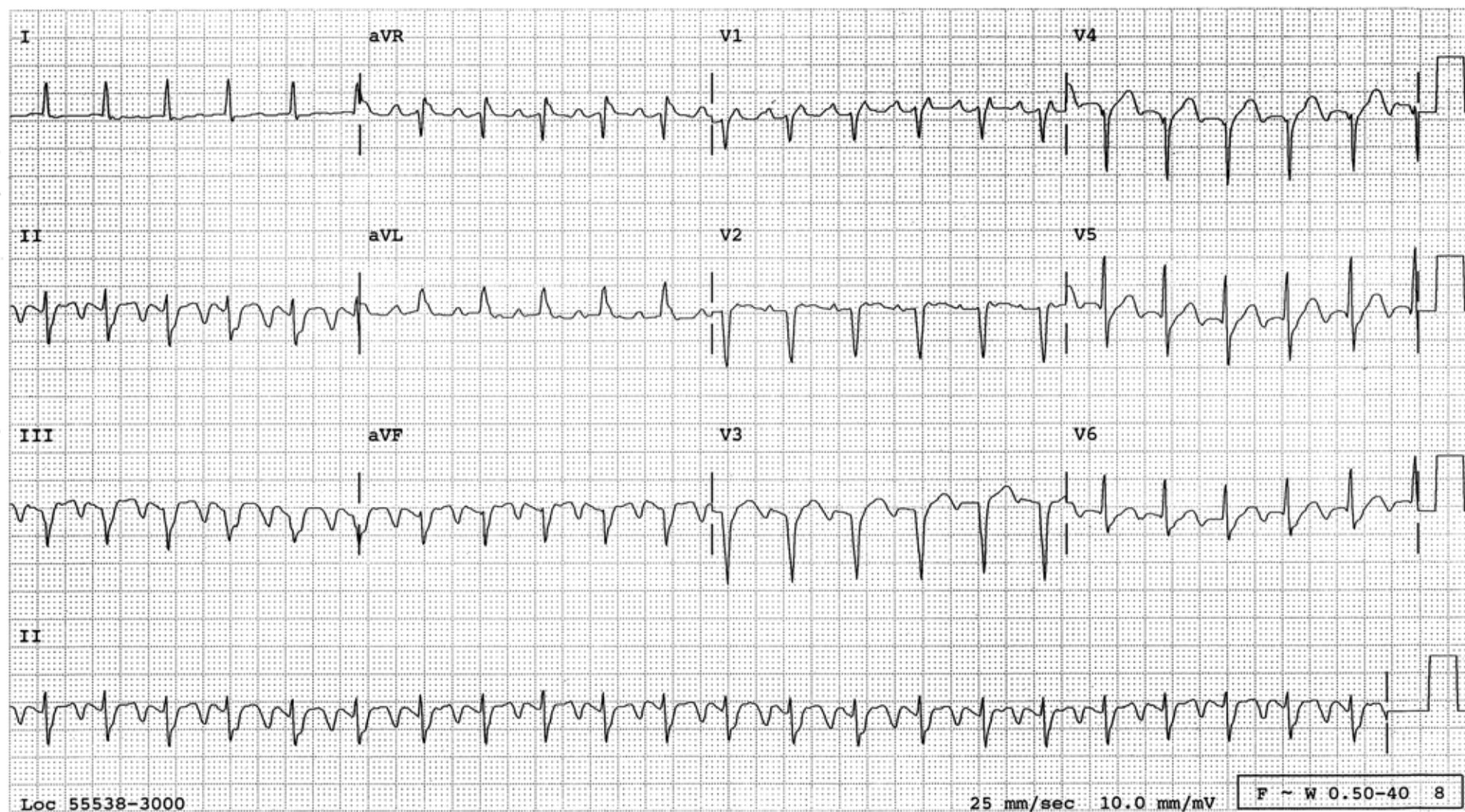
17-7 Sinus rhythm at a rate of 98/minute. Frequent atrial premature complexes are present. The P waves of these premature complexes are superimposed on the T waves.

- Dx: 1. Sinus rhythm
2. Multiple PACs



17-8 Normal sinus rhythm at 85/minute. T waves are symmetric and very tall. In some patients with STEMI, before the ST-segment is elevated, the T waves become like this and is called hyperacute T wave changes; more acute than acute!! Hyperkalemia also causes symmetric, tall T waves but the T waves are narrow, tented and pointed. Some people have tall T waves as a normal variant. In that case the T waves are not symmetric; the upstroke takes more time than the downstroke.

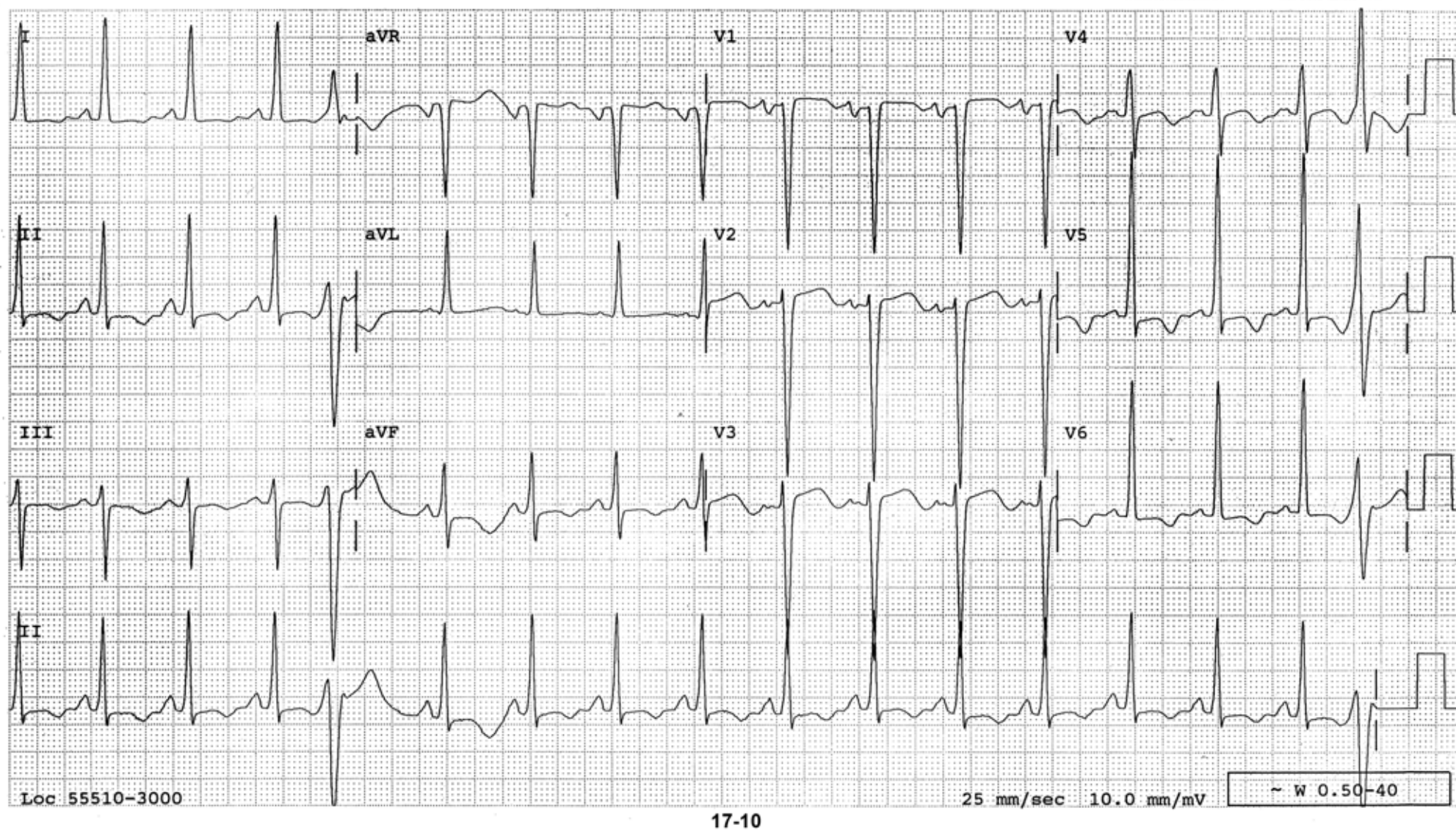
- Dx: 1. NSR
2. Hyperacute T wave changes



17-9

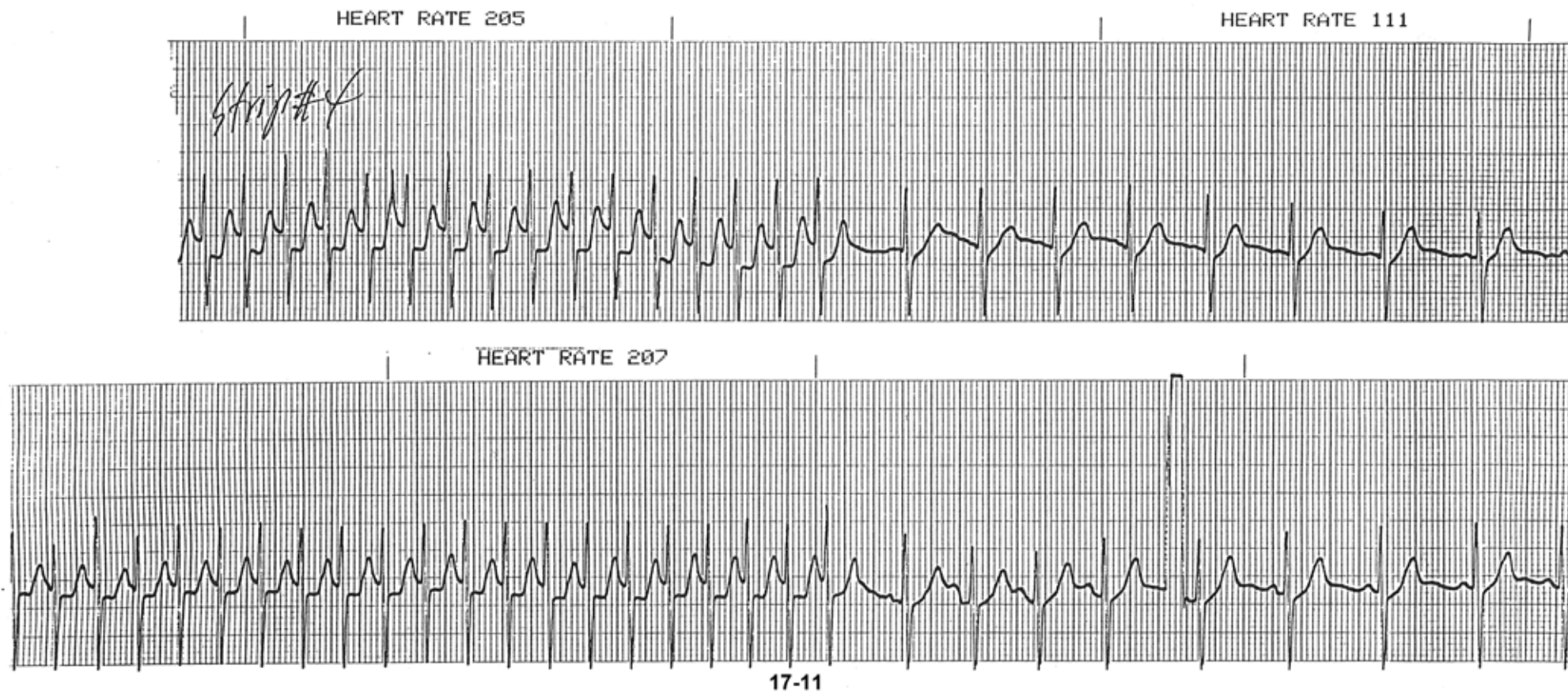
17-9 A narrow QRS tachycardia at a rate of 134/minute. The findings in V_2 suggest sinus tachycardia with one P wave in front of each QRS. However, V_1 reveals two atrial activities for each QRS. The atrial rate is 270/minute which is good for atrial flutter. This forces us to look for the flutter waves in inferior leads which are indeed there. Since every other flutter wave is deformed by the QRS, it is not readily recognizable. The mean QRS axis is shifted to the left. QS pattern in V_2 - V_4 indicates old anterior infarct.

- Dx:
1. Atrial flutter with 2:1 AV conduction
 2. LAFB
 3. Old anterior infarct



17-10 Normal sinus rhythm at a rate of 96/minute. Voltage criteria and ST-T changes for LVH are present. Broad and deeply negative P wave in V_1 indicates LAE. The P wave is tall in lead II measuring a little over 2.5 mm indicating RAE as well. Occasional ventricular premature complexes are present.

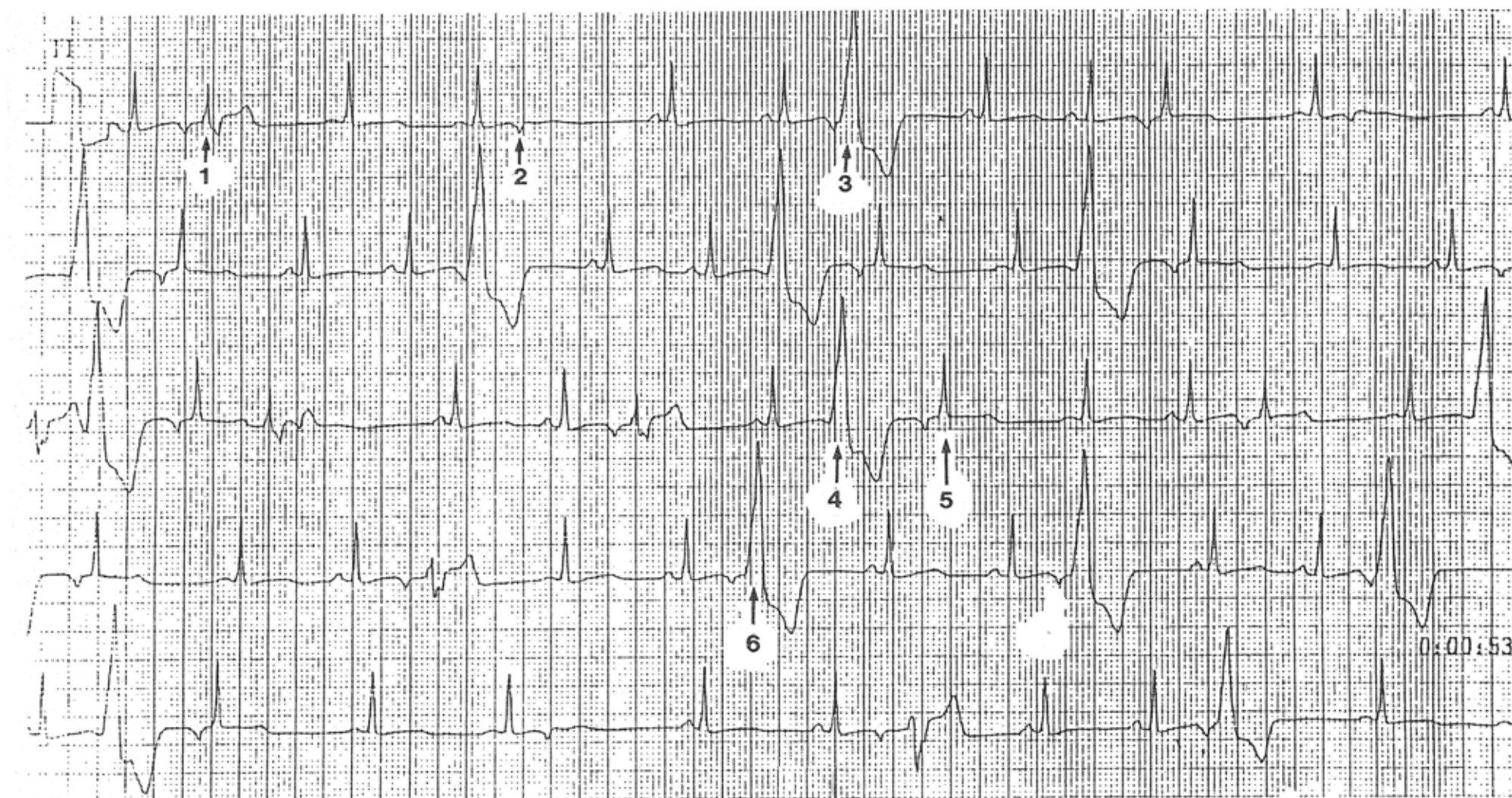
- Dx:*
1. NSR
 2. PVCs
 3. Biatial enlargements
 4. LVH



17-11 Two rhythm strips are present. The beginning of each of the rhythm strips reveals SVT at a rate of 210/minute. The rhythm spontaneously converts to sinus rhythm on both occasions. ST depression of about 5 mm is noted during the first strip. Myocardial ischemia induced by the tachycardia may cause this but tachycardia in the absence of ischemia can also produce this degree of ST depression. The fact that when the rhythm converts to sinus rhythm, the very first beat fails to reveal any ST depression indicating that this ST depression is due to tachycardia alone without ischemia, since ischemia cannot come and go from one beat to another.

- Dx:
1. Intermittent SVTs with spontaneous conversion
 2. Significant ST depression due to tachycardia alone without myocardial ischemia

17-12 Question: What are the waves pointed by the numbered arrows?

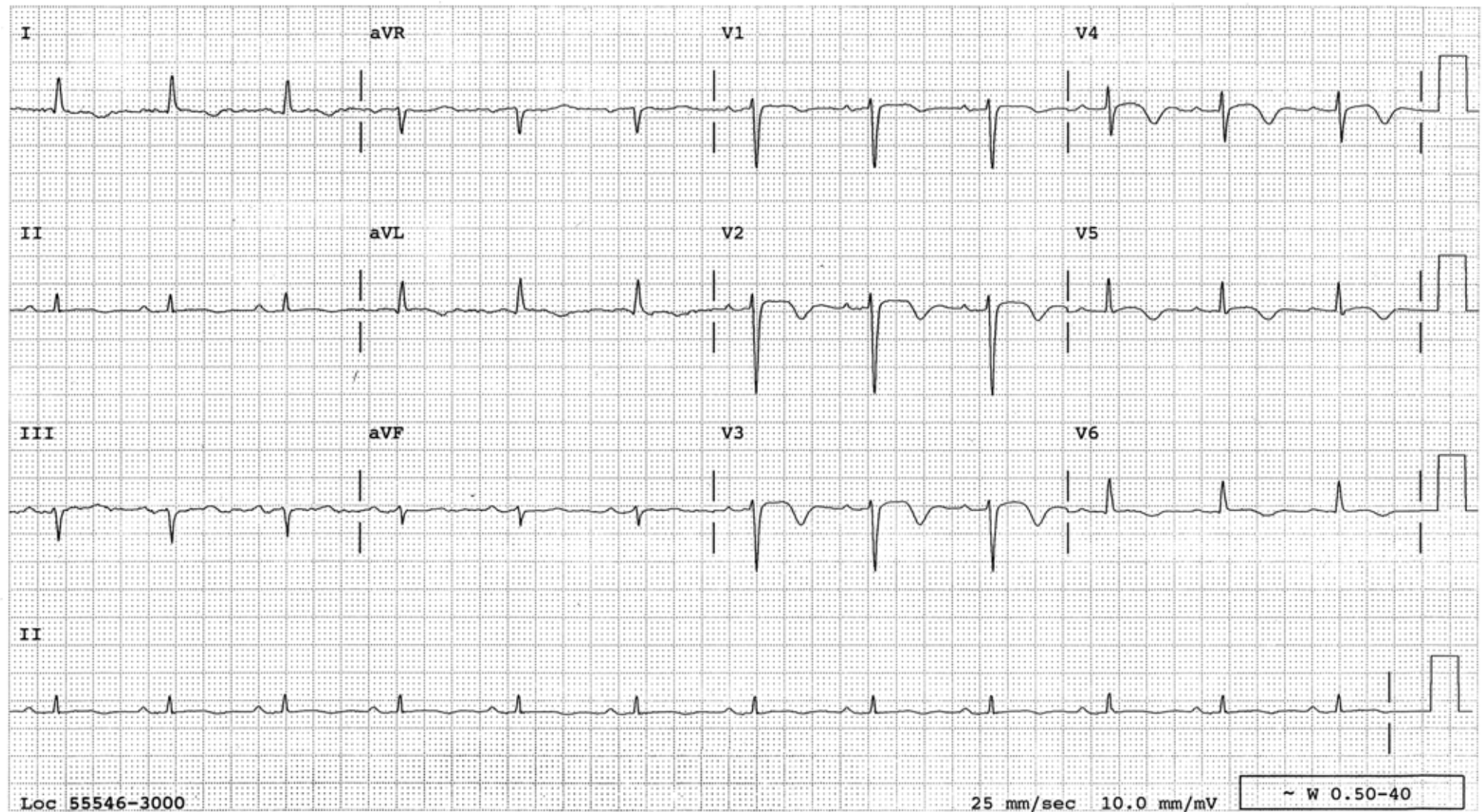


17-12

Answers:

1. PAC with aberrant conduction
2. Nonconducted PAC
3. PVC with a premature P wave in front
4. PVC
5. PAC with normal conduction
6. PVC with a premature P wave in front

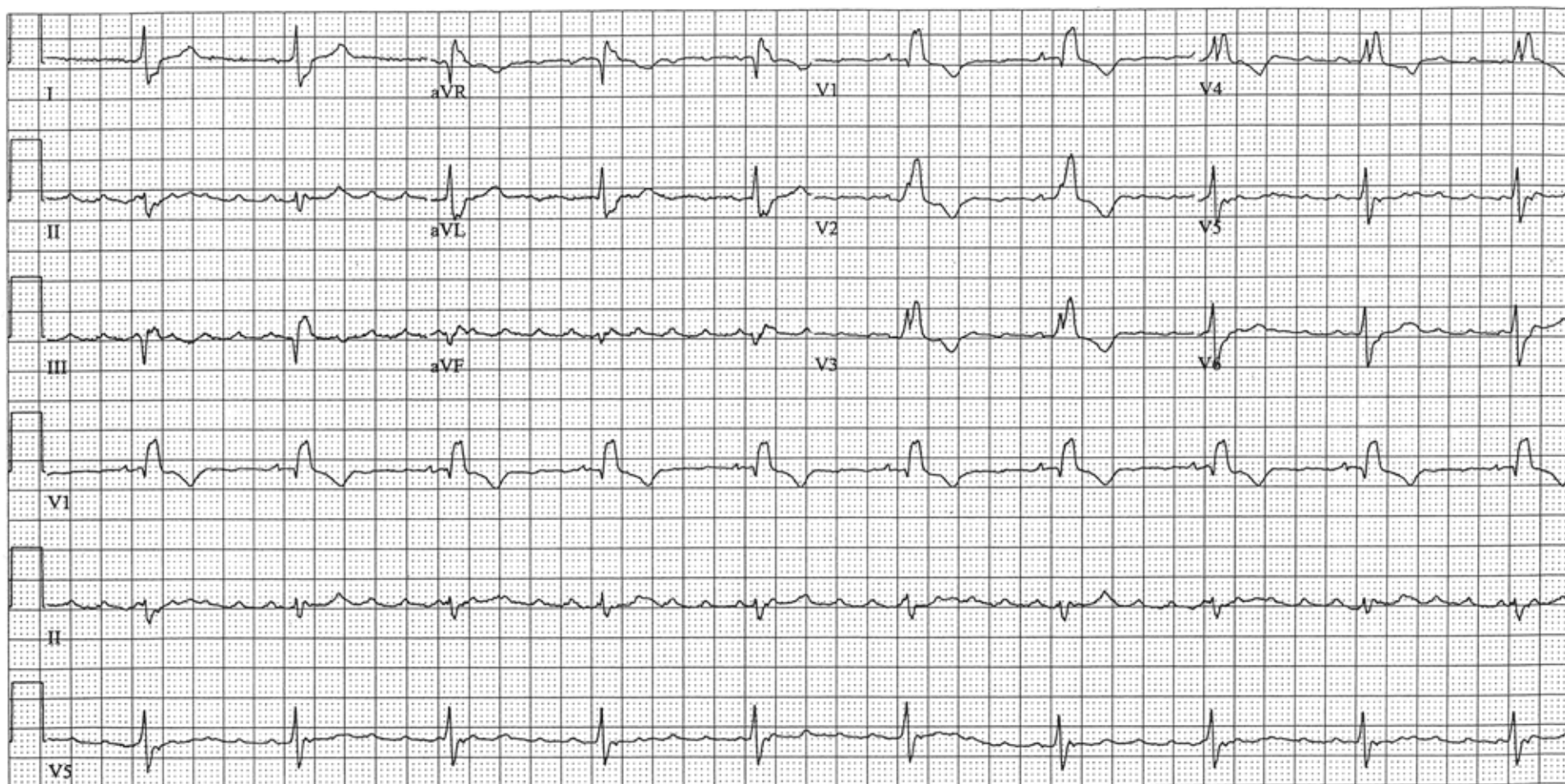
SECTION 18



18-1

18-1 NSR at a rate of 71/minute. T waves are symmetrically and deeply inverted in the precordial leads. The Q-T interval is prolonged. These findings may reflect either subendocardial ischemia or infarction, or stress cardiomyopathy.

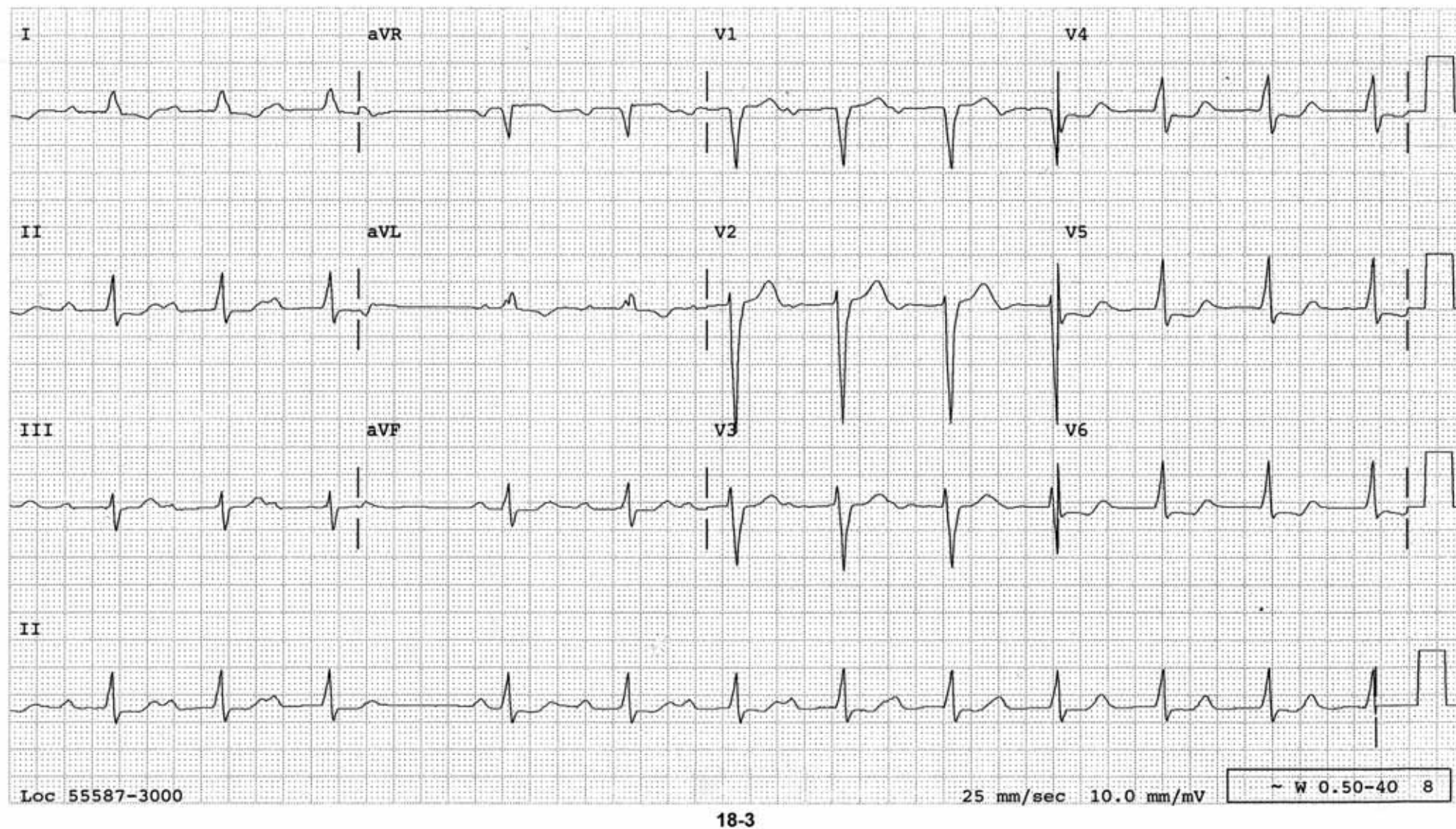
- Dx:
1. NSR
 2. Long Q-T interval
 3. ST-T changes suggestive of subendocardial ischemia or infarction, or stress cardiomyopathy



18-2

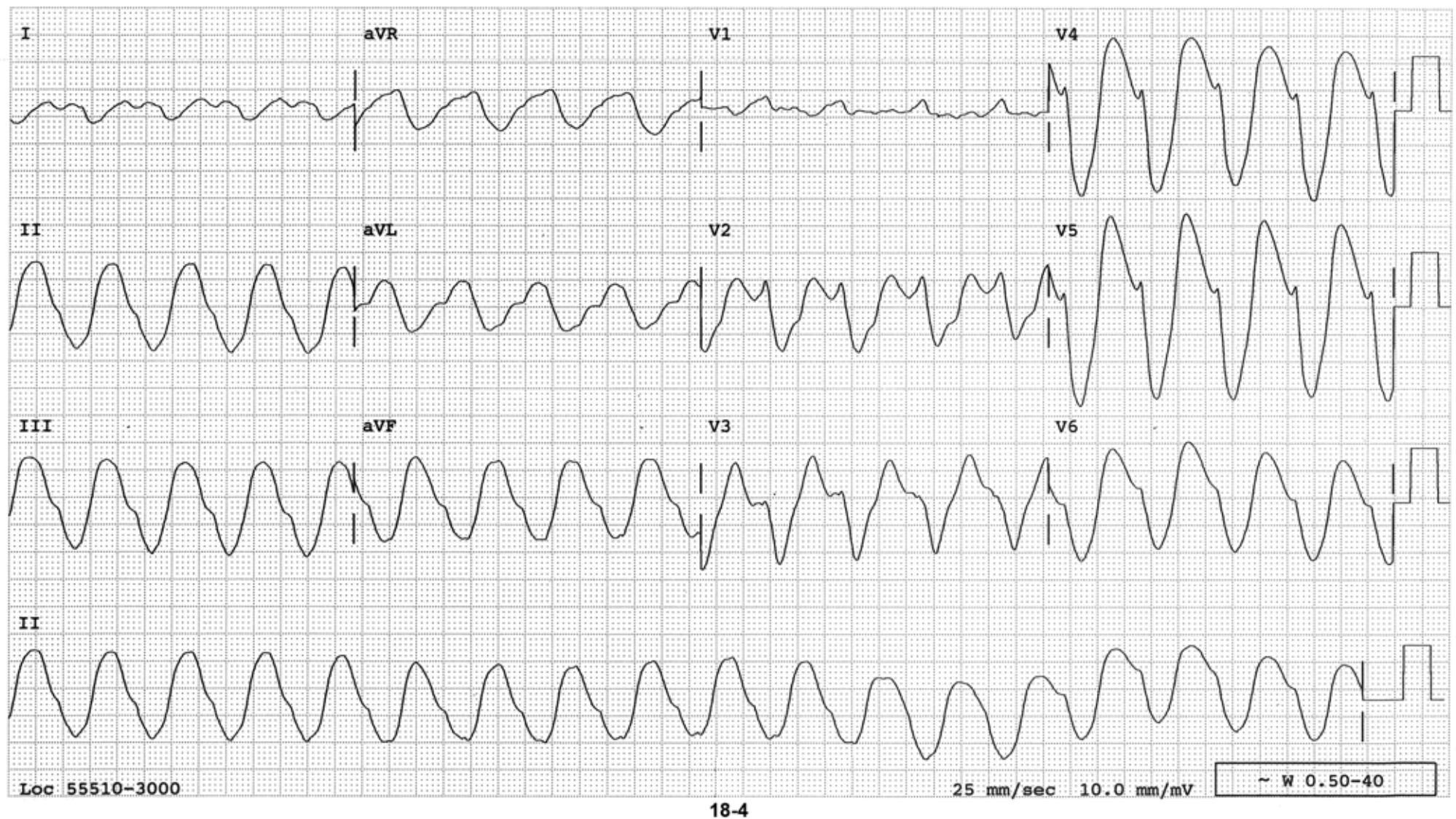
18-2 Regular QRS rhythm at 60/minute. QRSs are wide and typical of RBBB. NSR is recognizable in V_1 while typical atrial flutter waves are seen in other leads especially in the inferior leads. This tracing is from a patient who received a cardiac transplant. The recipient atrial cap is in atrial flutter while the donor heart is in sinus rhythm. If it weren't for the heart transplant, one could consider a condition called dissimilar atrial rhythm where one atrium is in one rhythm while the other atrium is in another. Another possibility that can be entertained is artifact from muscle tremor.

- Dx:
1. Atrial flutter of the recipient atrial cap and sinus rhythm of the donor heart in a patient with heart transplant
 2. RBBB



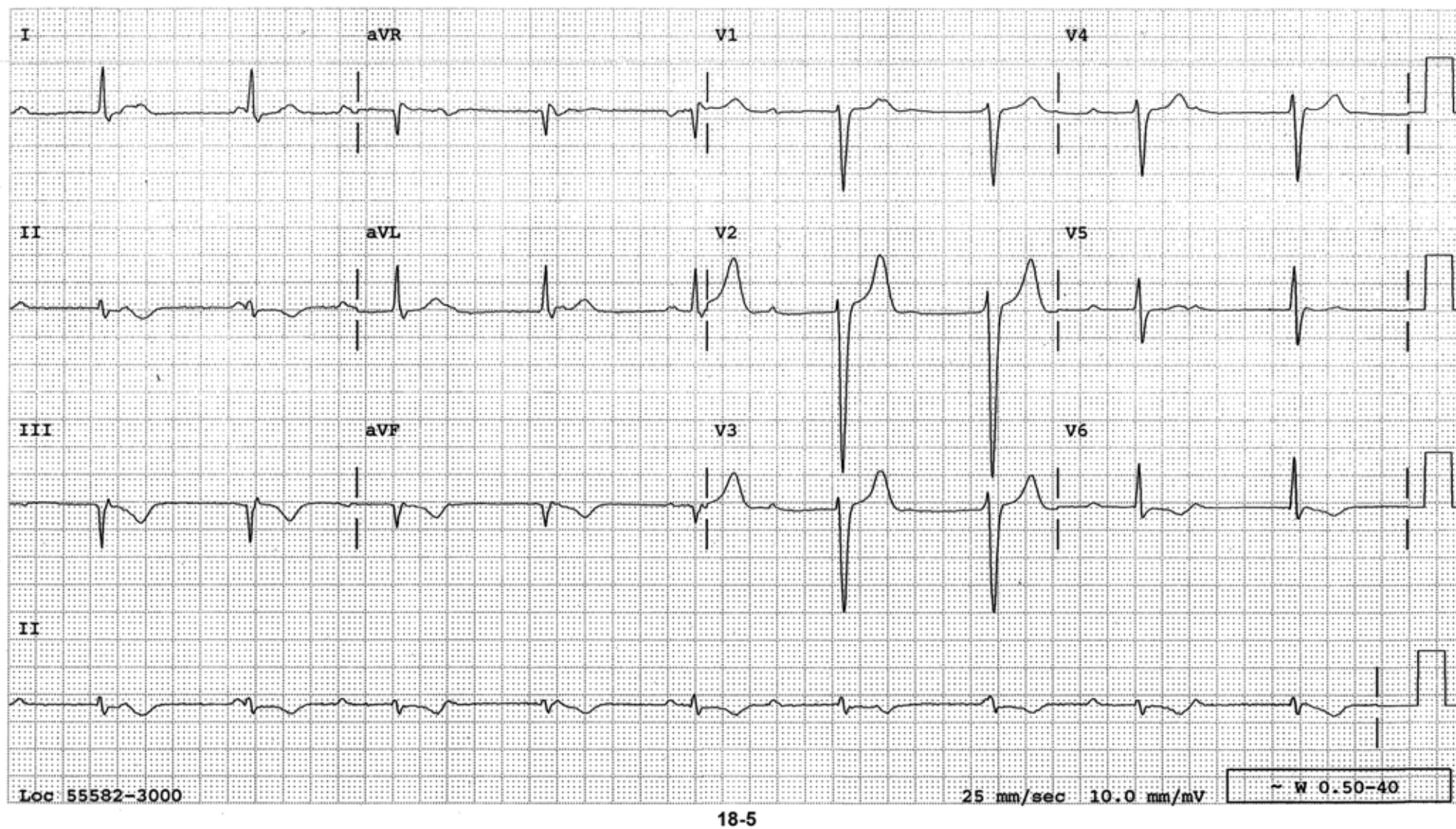
18-3 P waves occur regularly at a rate of 76/minute. The P-R interval is progressively lengthened until the fourth P wave is blocked. Then the AV Wenckebach cycle begins again with a progressively lengthening P-R interval. Toward the end of the tracing, P waves are superimposed upon the T waves but it would be difficult to be convinced that is what happened without seeing the earlier part of the tracing. The QRSs are wide measuring 117 milliseconds indicating either IVCD or incomplete LBBB.

- Dx: 1. Sinus rhythm with Type I 2° AV block
2. Incomplete LBBB



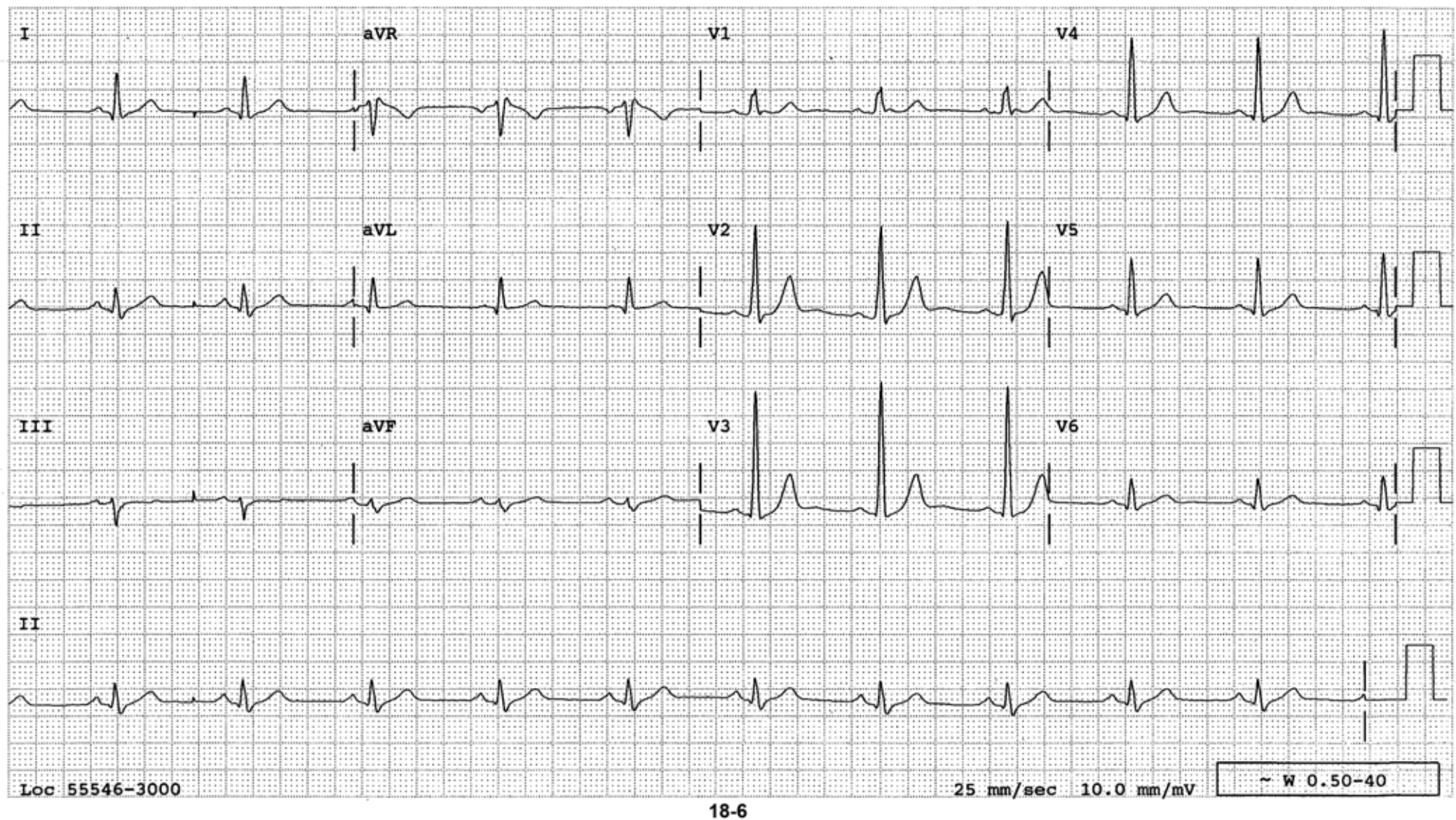
18-4 QRSs are very wide, too wide even for BBB or ventricular rhythm and are difficult to determine where it begins and where it ends. The T wave is somewhat prominent in V₅. This is an example of hyperkalemia. It is not diagnostic of the condition, but since hyperkalemia is a life-threatening situation and since it is easily correctable, it will be a great mistake not to consider the possibility. In fact the patient should be treated as such without waiting for the serum potassium level to become available, since if you were right, you might save the patient's life and if you were wrong, the treatment given for hyperkalemia (calcium, bicarbonate, glucose, and insulin) would not have caused any harm.

Dx: Findings highly suggestive of hyperkalemia



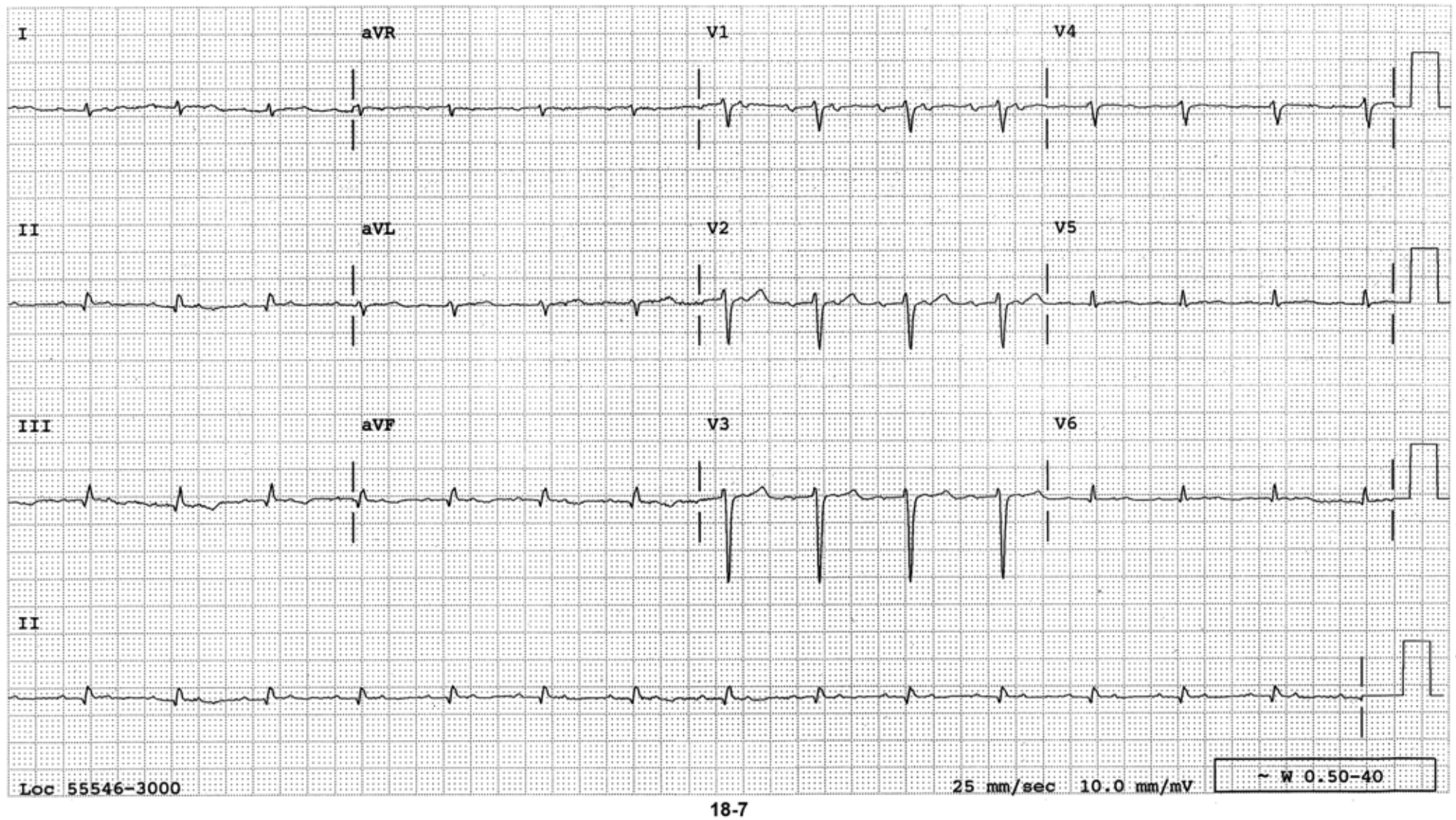
18-5 QRSs occur regularly at a rate of 55/minute. P waves also occur regularly at a rate of 75/minute but without any relationship to the QRSs indicating complete AV block. The QRS width is not wide enough to make one think of ventricular escape rhythm and most likely this is AV junctional escape rhythm. Q waves with T wave inversion in inferior leads indicate recent inferior MI which may be the cause of this complete AV block.

- Dx:
1. Complete AV block and junctional escape rhythm
 2. Inferior infarct, recent



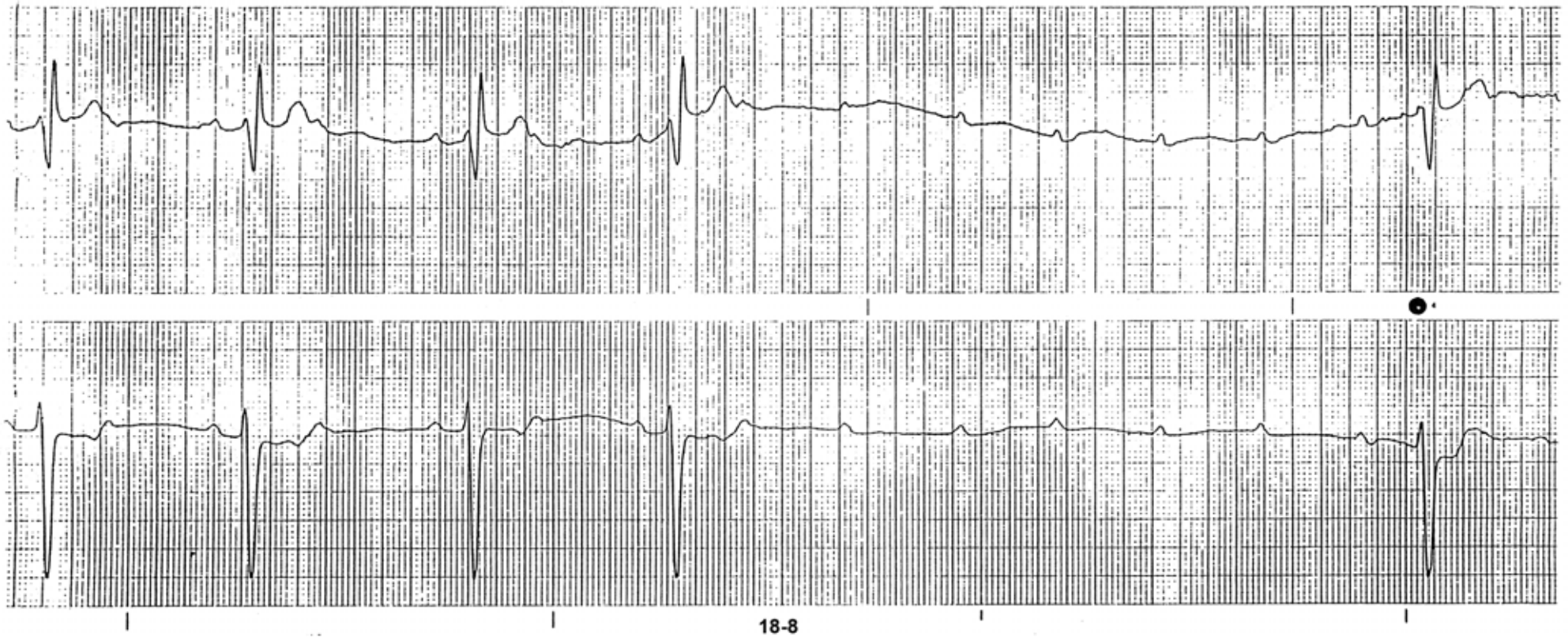
18-6 Normal sinus rhythm at a rate of 65/minute. QRSs are entirely positive in the right precordial leads, which is distinctively abnormal. The differential diagnosis of tall R waves in the right precordial leads include RVH, posterior MI, and some cases of WPW syndrome and reversed precordial leads. RVH should accompany RAD and deep S waves in the left precordial leads which are not the case in this tracing. The P-R interval is not short in any leads and there are no convincing delta waves ruling out WPW syndrome. This is an example of old posterior MI. Many times, posterior MI occurs as a part of inferoposterior or posterolateral MI. However, occasionally isolated posterior MI can occur as in this patient. If it is an acute posterior infarction, the ST-segment will be depressed as a reciprocal change of ST elevation in the posterior wall. Since the ST-segment is not depressed, this is an old infarct.

- Dx: 1. NSR
2. Old posterior infarct



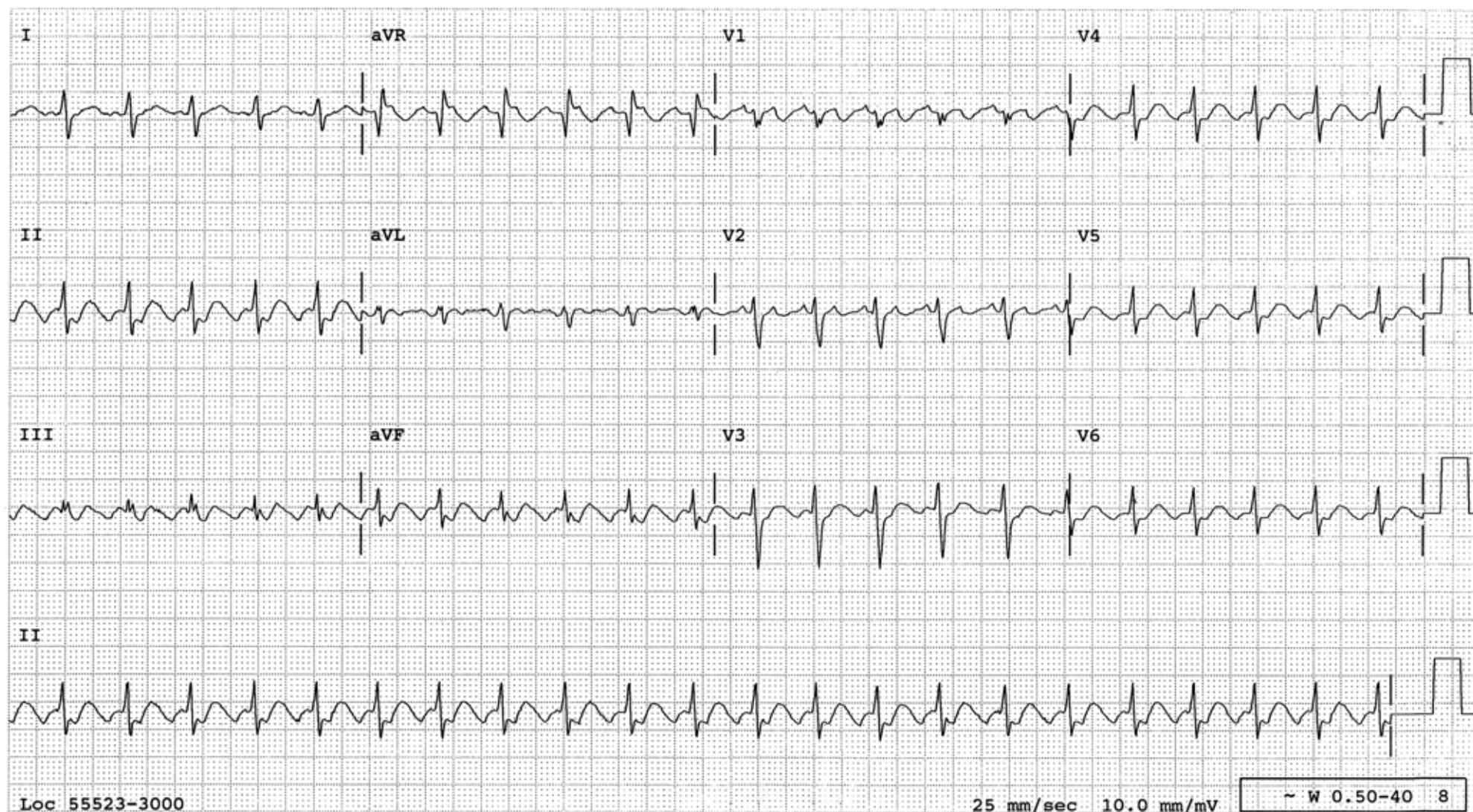
18-7 A regular rhythm at a rate of 90/minute. Two atrial activities are present between the QRSs in V_1 at a rate of about 180/minute. There is no definite sawtooth pattern in the inferior leads and this is an example of atrial tachycardia with 2:1 AV conduction rather than atrial flutter. Atrial tachycardia with a variable AV conduction should raise the possibility of digitalis toxicity. The QRS voltage is low in the limb leads.

- Dx:*
1. Atrial tachycardia with 2:1 AV conduction
 2. Low QRS voltage
 3. Poor R wave progression



18-8 A rhythm strip of two channels taken simultaneously revealing 2:1 AV block initially followed by many blocked P waves indicating high grade AV block. The QRS is wide indicating IVCD. Twelve-lead ECG would be interesting to see whether the patient has BIFB (complete RBBB and left anterior or left posterior fascicular block). If that is the case in a patient with a high grade AV block, artificial pacemaker is indicated.

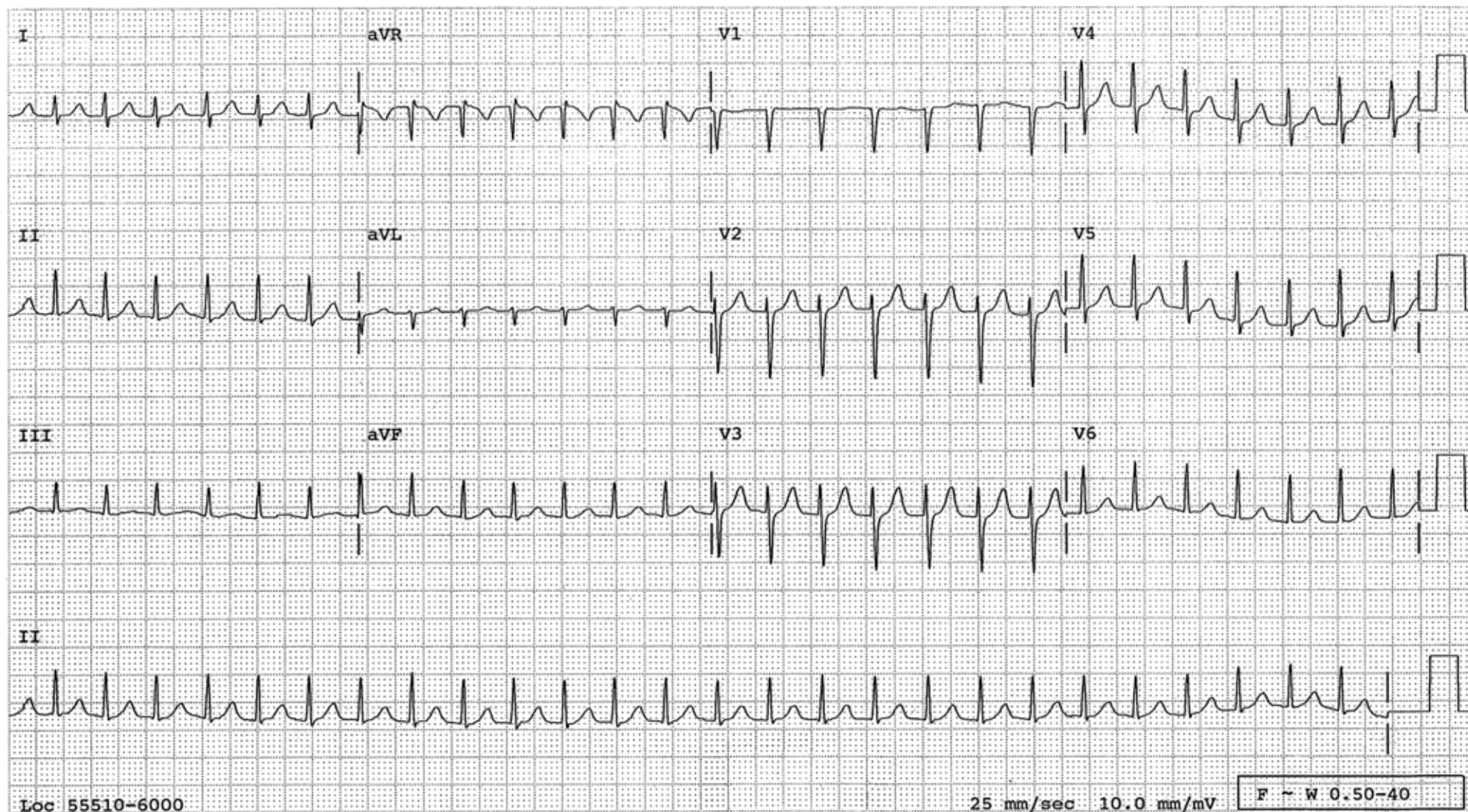
Dx: High grade AV block



18-9

18-9 A typical example of atrial flutter with 2:1 AV conduction. The sawtooth pattern of atrial flutter can be readily appreciated in the inferior leads. V_2 reveals two atrial activities for each QRS, further supporting the diagnosis. The mean QRS axis is shifted to the right and S waves remain prominent in the precordial leads which may be due to obstructive pulmonary disease. The ST-segment cannot be assessed on account of flutter waves.

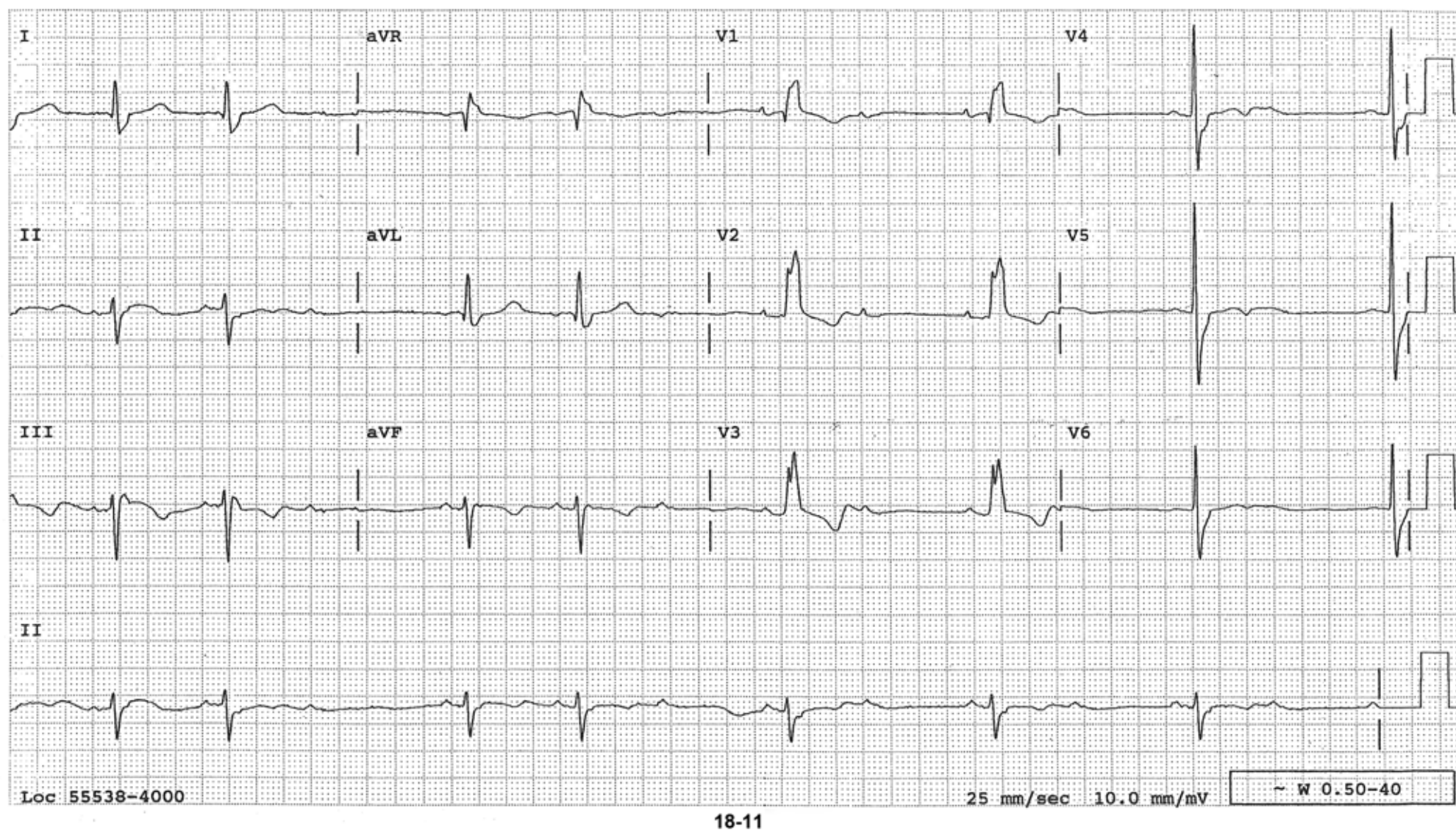
- Dx:
1. Atrial flutter with 2:1 AV conduction
 2. RAD
 3. Consider COPD



18-10

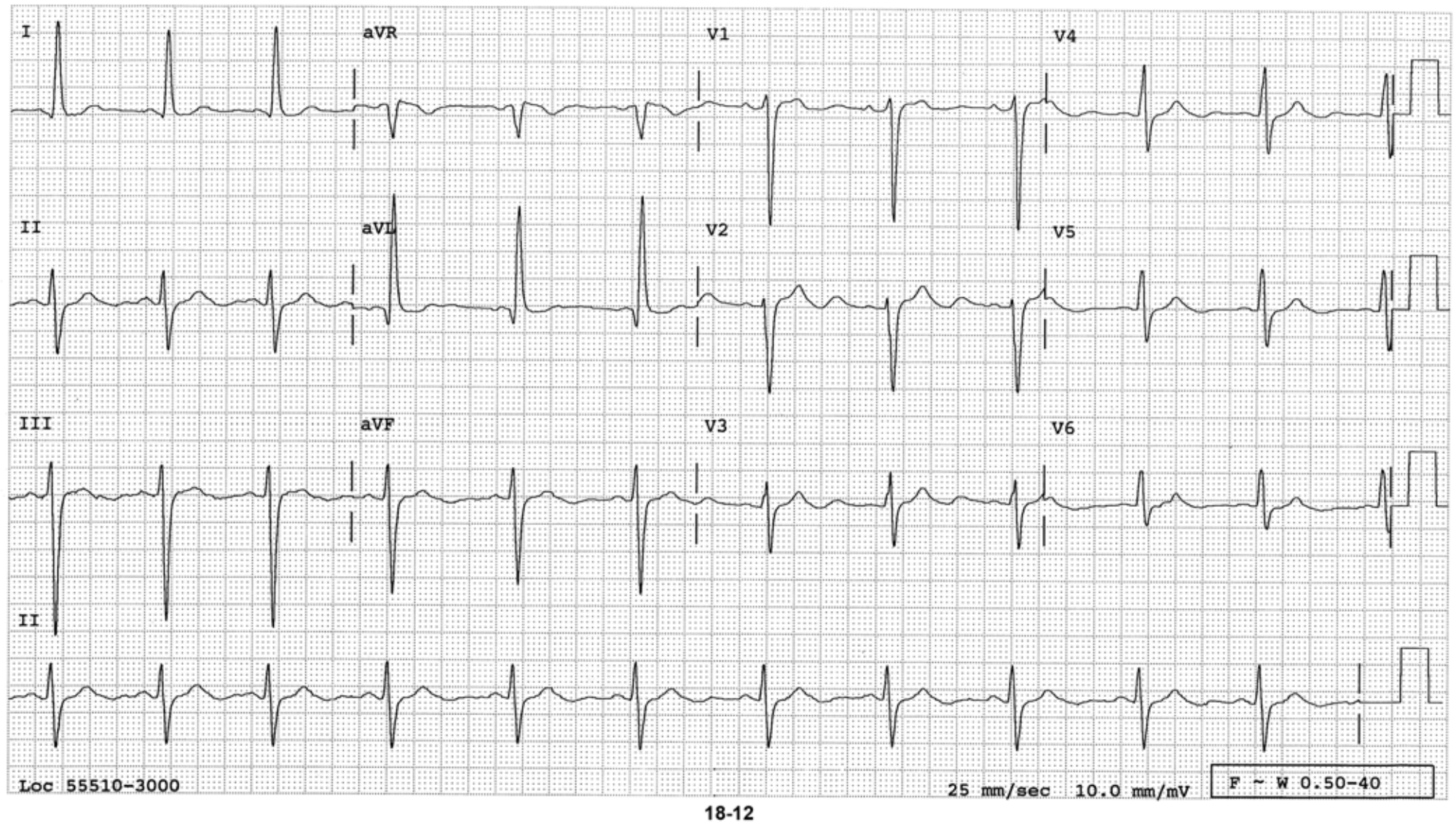
18-10 A narrow QRS tachycardia at a rate of 162/minute. No definite P waves can be identified. For not being able to be more specific, this will be called generically SVT which is made of either ectopic atrial tachycardia, AV junctional tachycardia, AV junctional reentrant tachycardia (~60% of SVTs), or AV reentrant tachycardia utilizing accessory pathway (~30% of SVTs). An electrophysiologic study is often necessary to make the specific diagnosis.

Dx: SVT



18-11 Normal sinus rhythm at a rate of 80/minute. Many P waves are blocked. When the P waves are conducted to the ventricles, the P-R interval remains fixed indicating that this is Type II 2° AV block. The patient has complete RBBB and LAFB (BIFB) which also support that the location of the block is most likely below the His bundle. A permanent pacemaker is indicated.

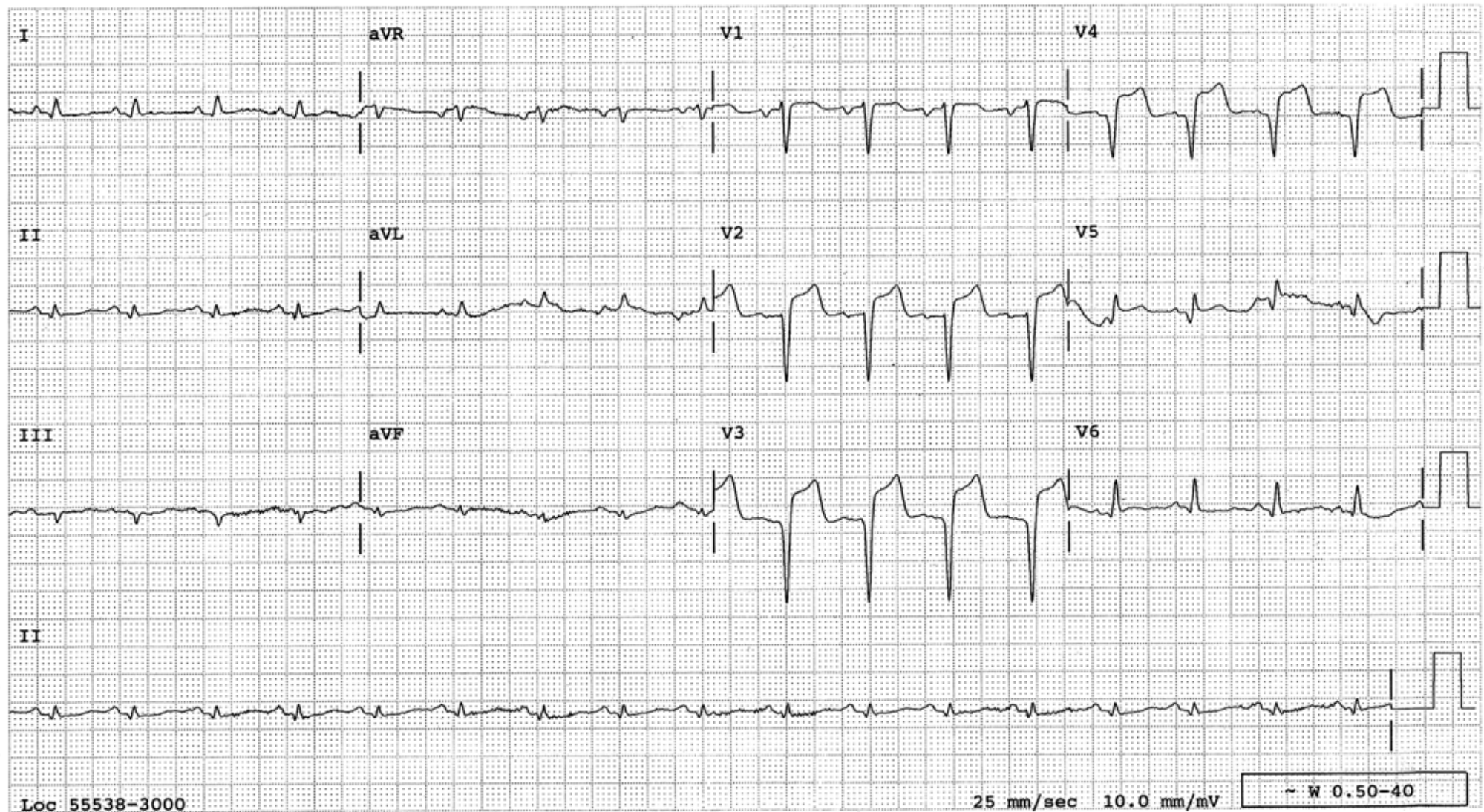
- Dx:
1. NSR
 2. Type II 2° AV block
 3. Bifascicular block (RBBB and LAFB)



18-12 Normal sinus rhythm at a rate of 68/minute. The QRS axis is shifted to the left, indicating LAFB. The QRS width measures 122 milliseconds, reflecting IVCD. The QRS voltage of 2 mV in aVL is very good for LVH. Prominent U waves are present in the right precordial leads, reflecting either LVH or hypokalemia.

- Dx:
1. NSR
 2. LAFB with ICVD
 3. LVH
 4. Prominent U waves, either secondary to LVH or hypokalemia

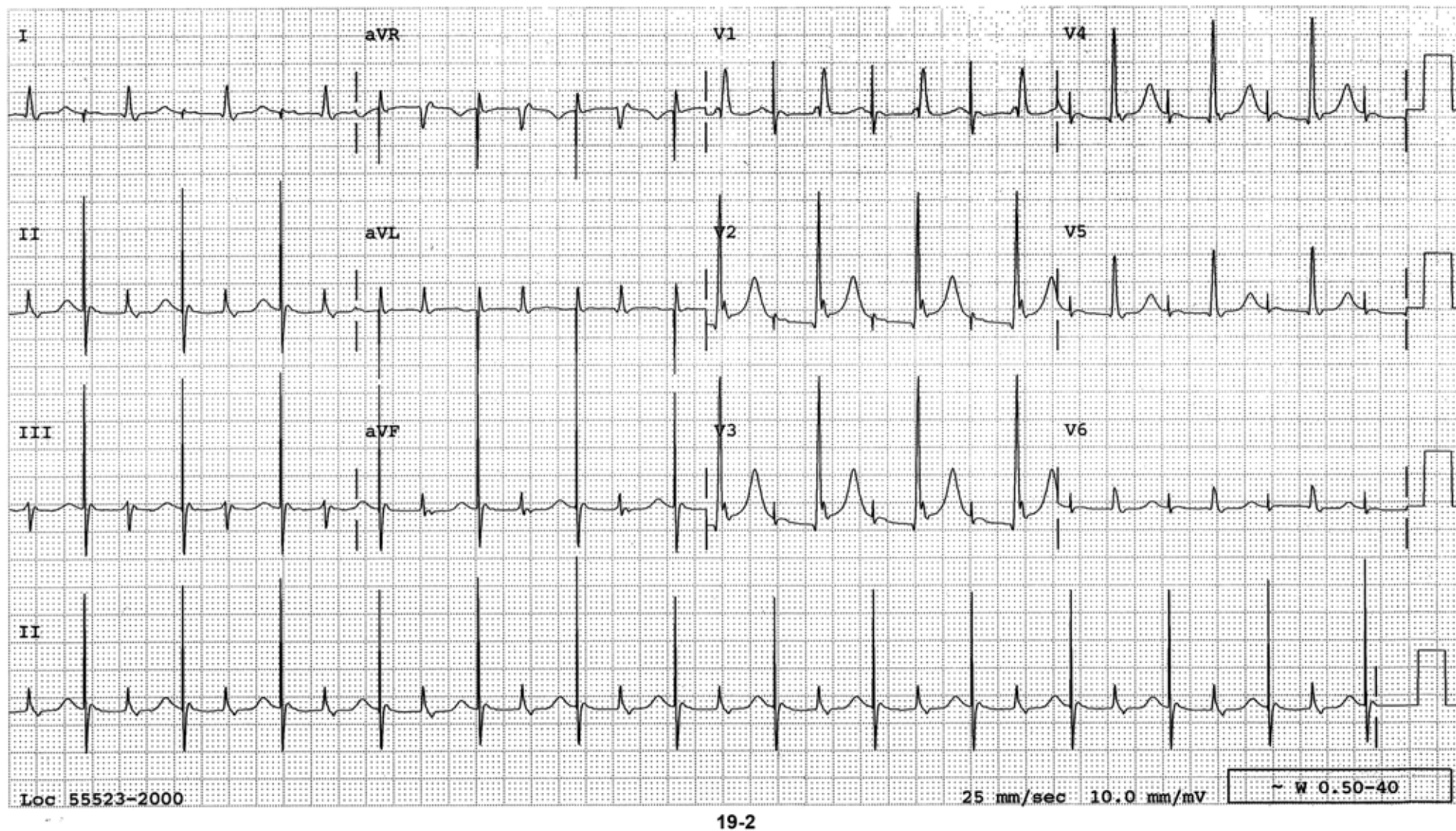
SECTION 19



19-1

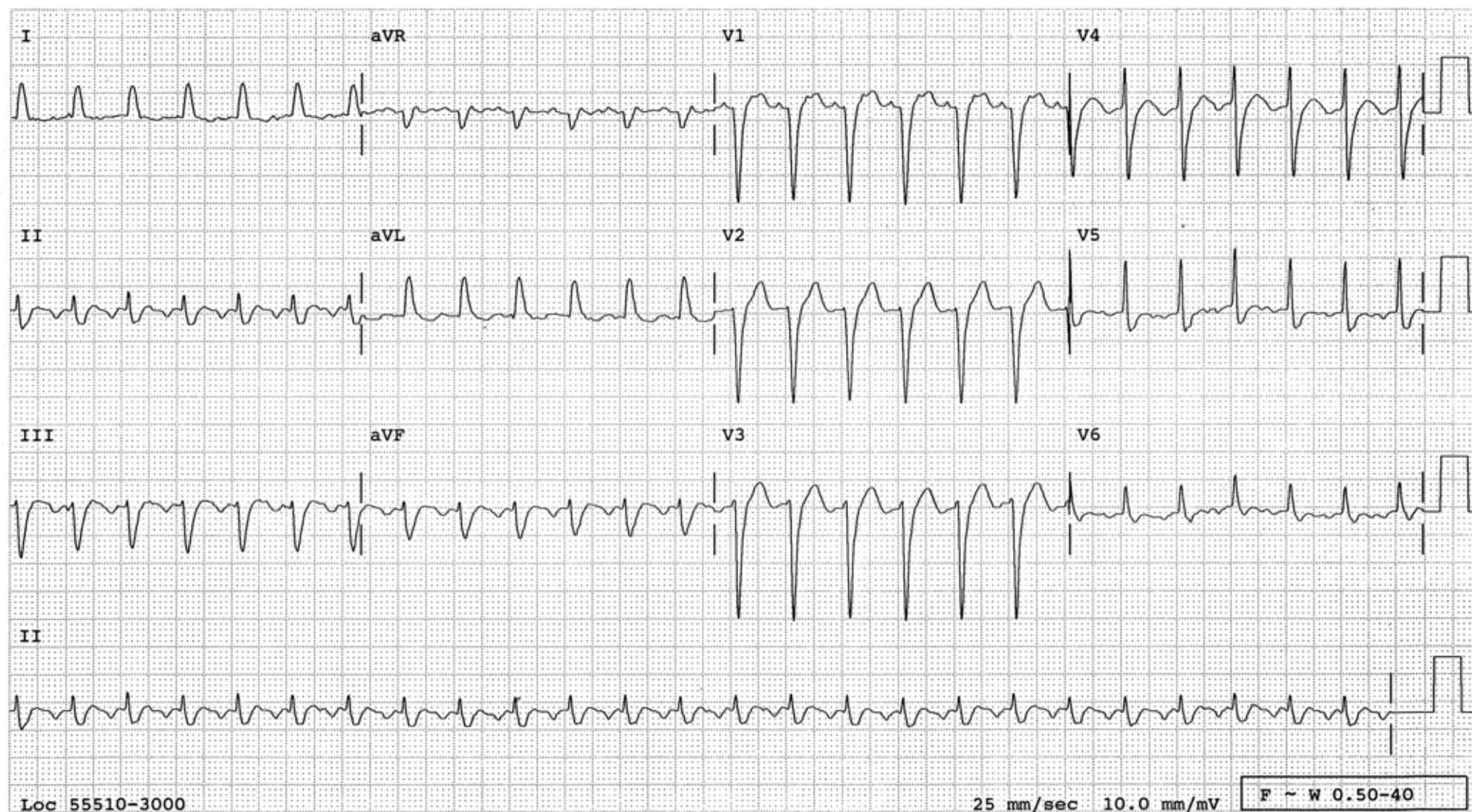
19-1 Sinus tachycardia at a rate of 102/minute. The QRS voltage is low in the limb leads. The P wave is mostly negative in V_1 indicating LAE. Q waves with a significant ST elevation in the precordial leads indicate acute extensive anterior MI. QS pattern in lead III with small Q waves in leads II and aVF suggest old inferior MI as well.

- Dx:*
1. Sinus tachycardia
 2. LAE
 3. Low QRS voltage in the limb leads
 4. Probably old inferior infarct
 5. Acute extensive anterior infarct



19-2 Artificial pacemaker spikes are readily recognizable. The pacemaker is pacing the atrium. After a long delay, the patient's own QRS occurs which has RBBB configuration.

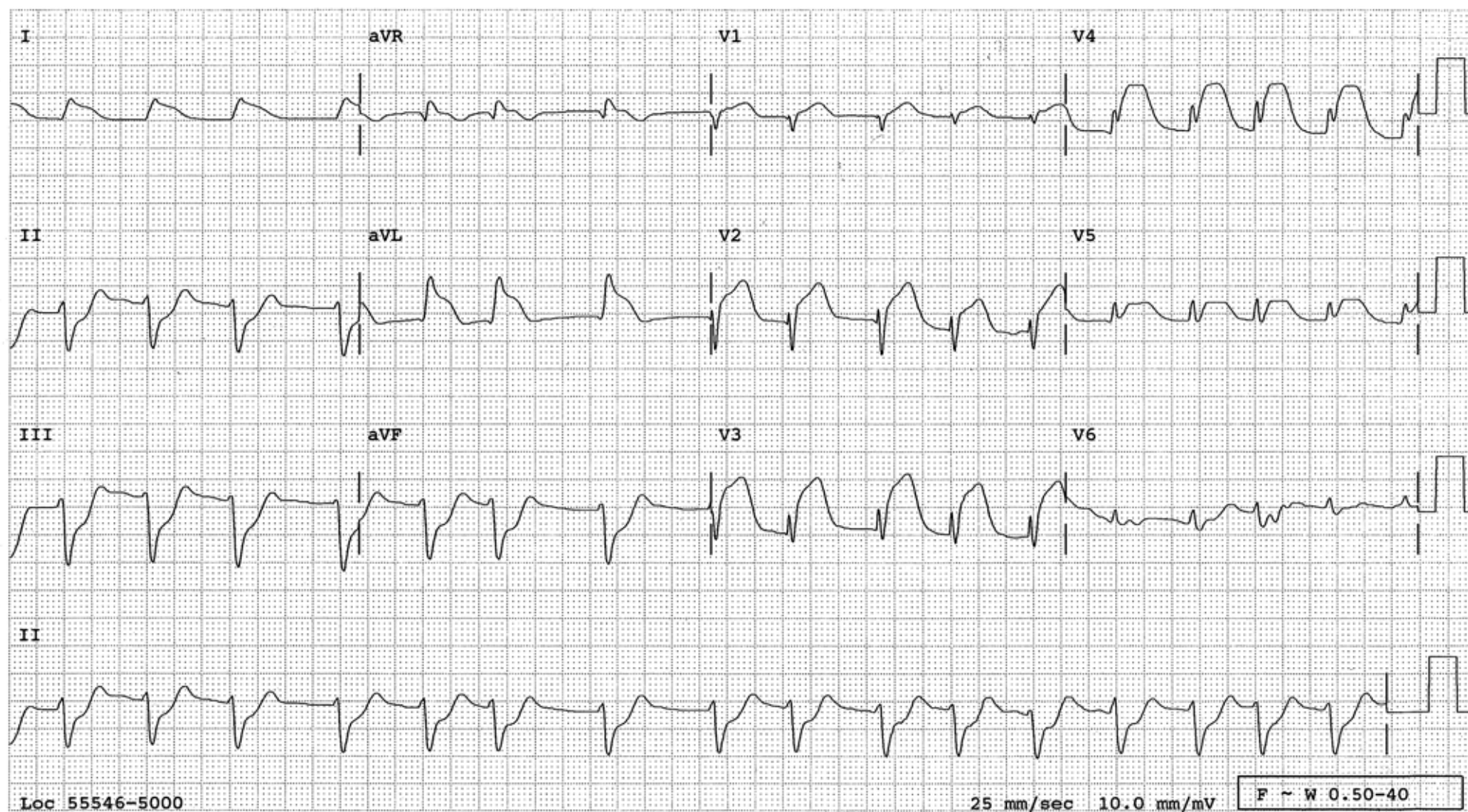
- Dx:*
1. Atrial paced rhythm
 2. 1° AV block
 3. RBBB



19-3

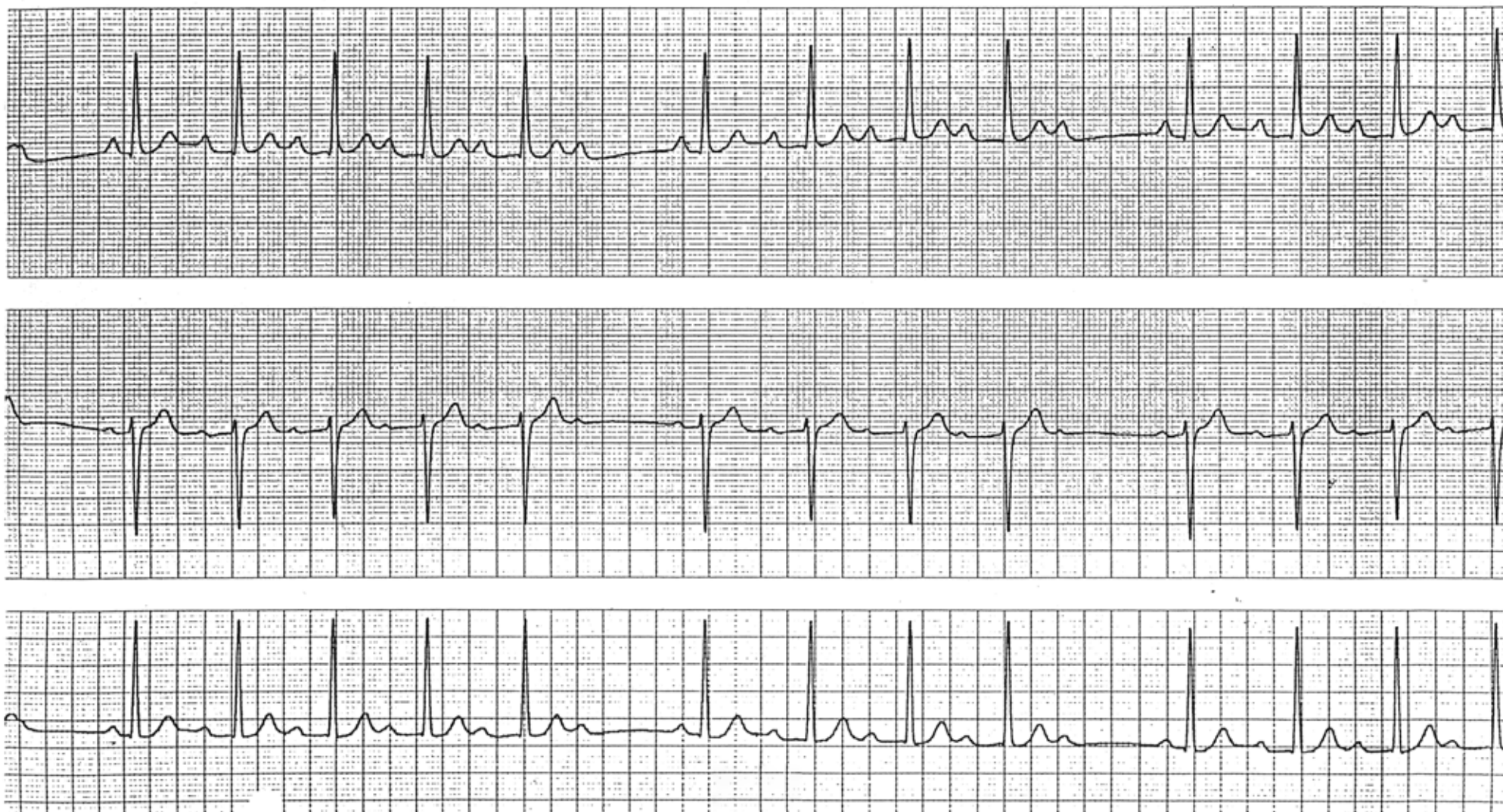
19-3 A regular tachycardia at a rate of 150/minute. QRSs are slightly wide and measures just under 120 milliseconds. In V₁, one "P" wave is clearly visible. The blip on the upstroke of the T wave is another atrial activity indicating a regular atrial rhythm at a rate of 300/minute. The atrial rate of 300/minute is diagnostic of atrial flutter. With this information, the flutter waves become more evident in the rhythm strip of lead II.

- Dx: 1. Atrial flutter with 2:1 AV conduction
2. IVCD



19-4 Irregularly irregular rhythm with no visible P waves indicates atrial fibrillation. Significant, coved ST elevation in the precordial leads as well as in leads I and aVL indicate acute extensive anterior MI. Lead I and aVL represents high lateral wall which often is perfused by diagonal branch which takes off very proximally in LAD. Therefore infarction pattern involving precordial leads and I and aVL means the culprit lesion is in the proximal LAD. If leads I and aVL are not involved, the lesion is in the LAD not proximal. If only leads I and aVL are involved without precordial leads, the lesion is in the diagonal branch. The ST depression in the inferior leads is the reciprocal change of the ST elevation in aVL.

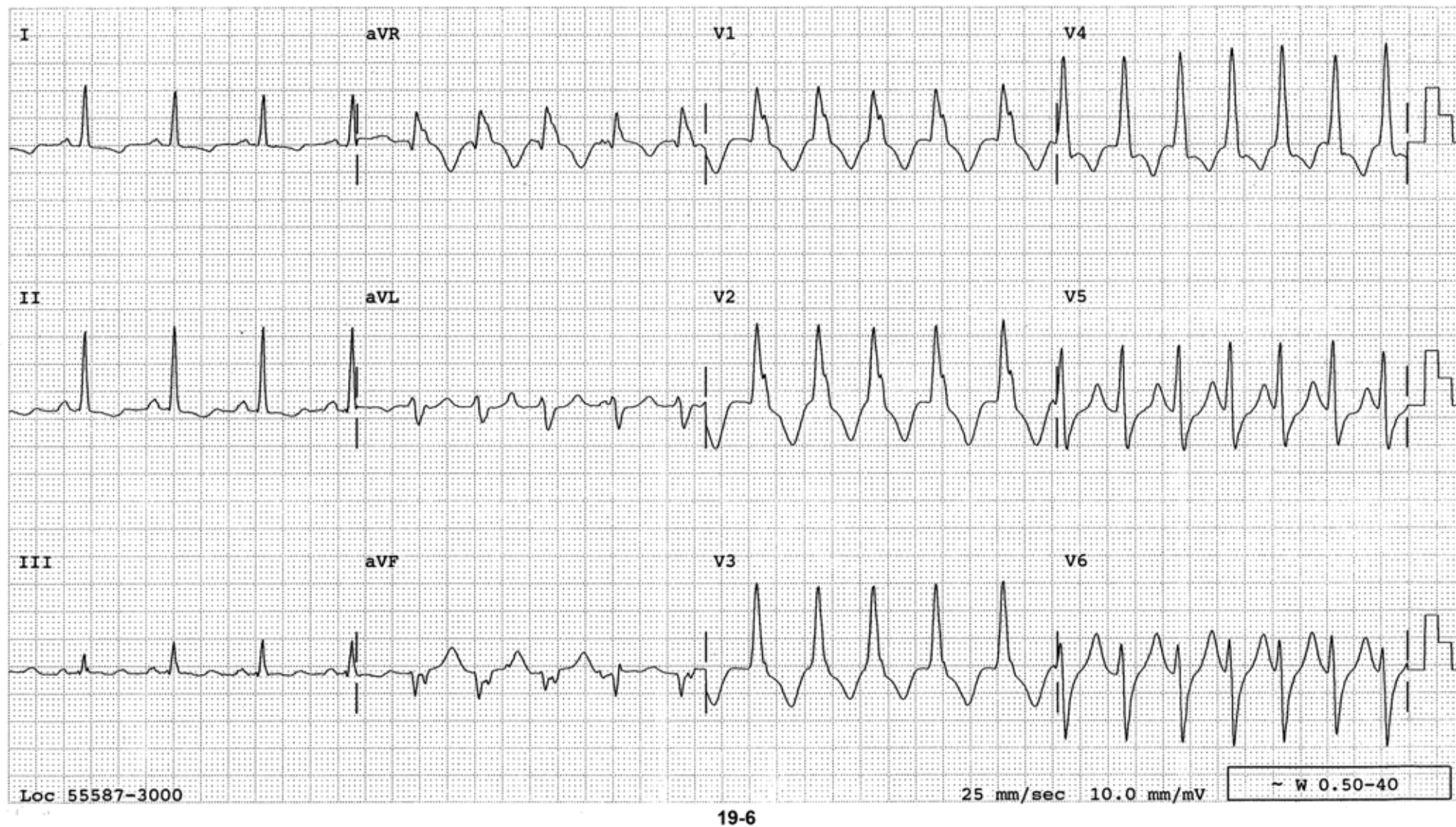
- Dx: 1. Atrial fibrillation with a ventricular response of 85/minute
2. Acute, extensive anterior infarct



19-5

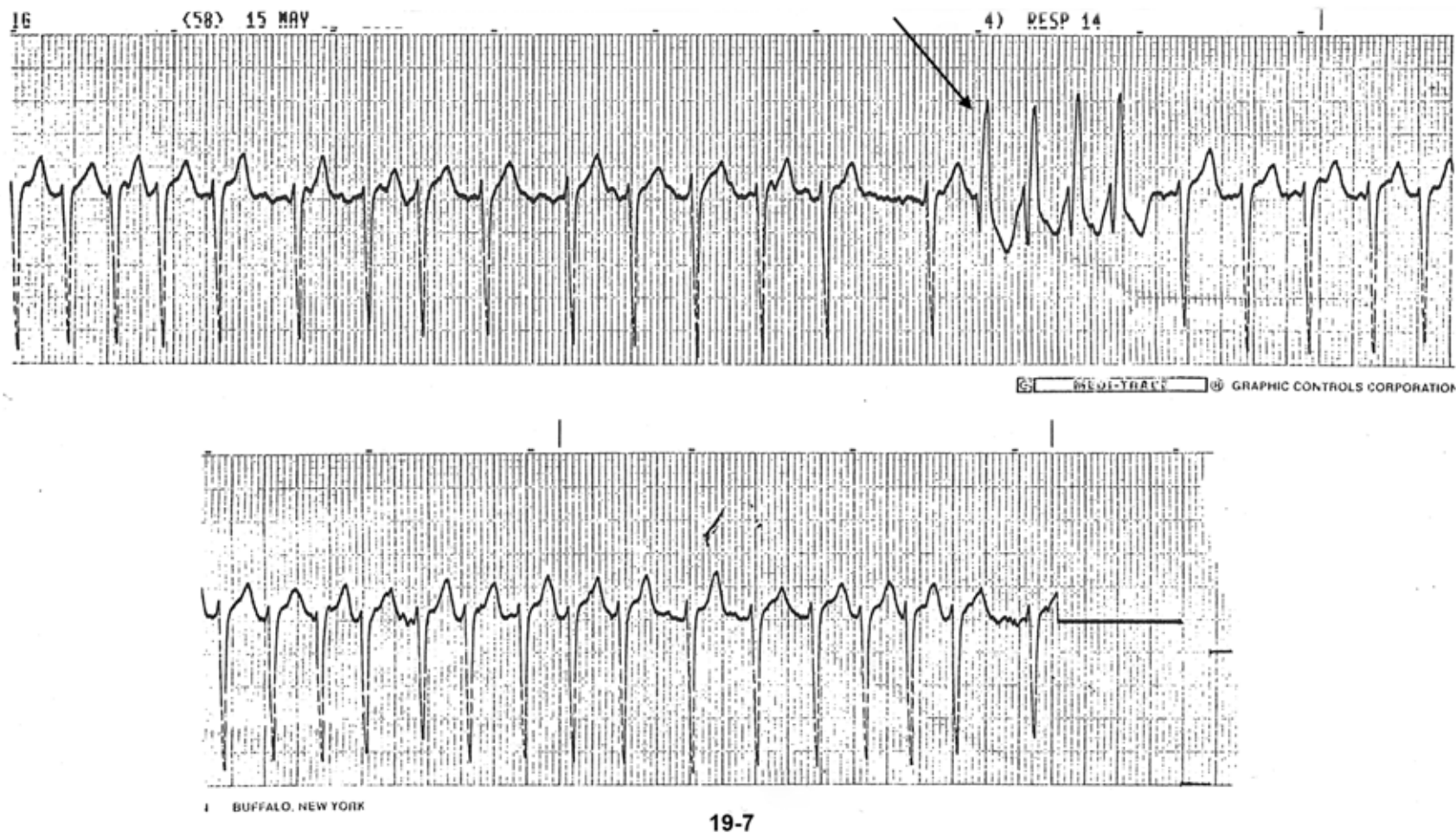
19-5 In these three channel rhythm strips, P waves occur regularly at a rate of about 85/minute. Occasionally, the P waves failed to result in a QRS. Prior to that, the P-R interval progressively lengthens; a typical Type I 2° AV block.

Dx: Type I 2° AV block (AV Wenckebach phenomenon)



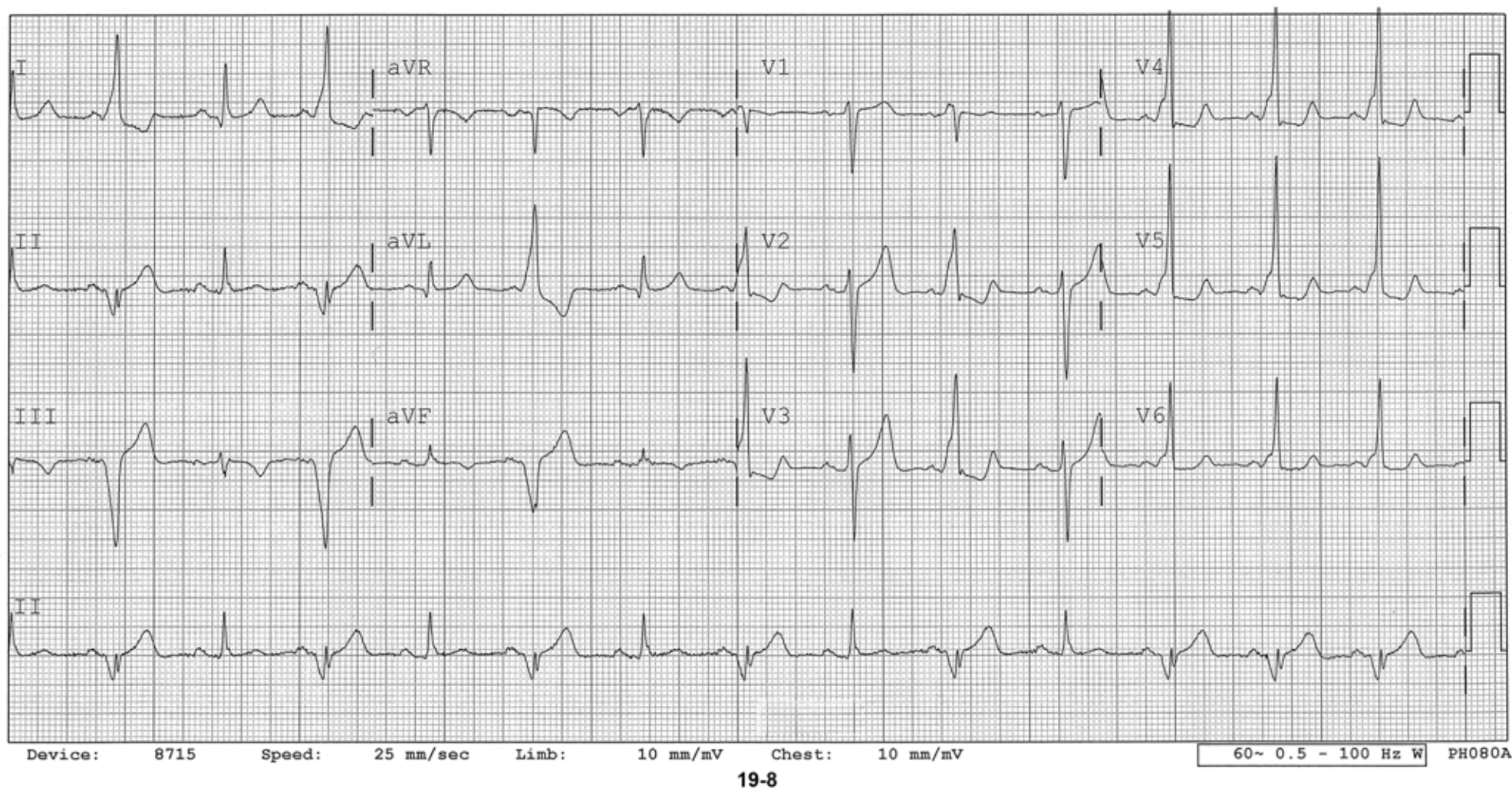
19-6 A sinus rhythm is present in the beginning of the tracing which is followed by a wide QRS tachycardia at a rate of 140/minute. The first complex of this wide QRS tachycardia does not have a P wave in front of it, proving that complex originated from the ventricle; hence this is a run of VT.

Dx: Sinus rhythm followed by VT



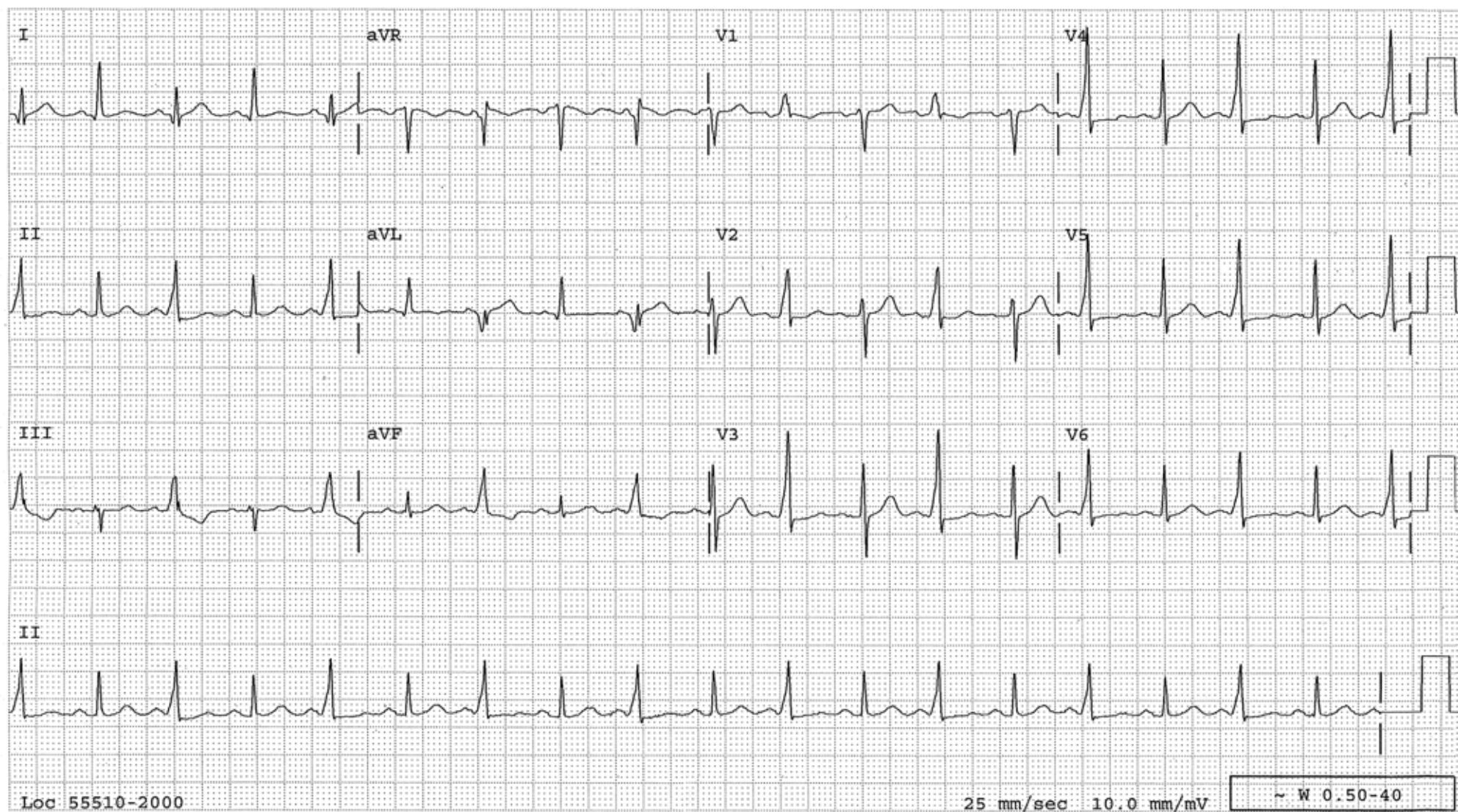
- 19-7 Irregularly irregular rhythm with no organized P waves indicates atrial fibrillation. Toward the end of the first rhythm strip, there are four wide QRS complexes. They have RBBB configuration favoring aberrant conduction rather than ventricular complexes. The preceding R-R interval is long suggesting Ashman's phenomenon which states that the complex which follows a longer preceding RR cycle is more likely to be aberrantly conducted.

Dx: Atrial fibrillation with a short run of aberrantly conducted complexes (Ashman's phenomenon)



- 19-8 Every other QRS complex is wide and different compared to other QRSs and occurs slightly prematurely, raising a possibility of ventricular bigeminy during NSR. But then, these beats have a short P-R interval and a slurred upstroke raising a possibility of preexcitation too. The QRS complexes in V_4 - V_6 settle the issue. Now every beat is preexcited, confirming that it is preexcitation of alternate beats. Preexcitation can be intermittent; only every other beat is preexcited as in this case, several beats at a time or several days at a time.

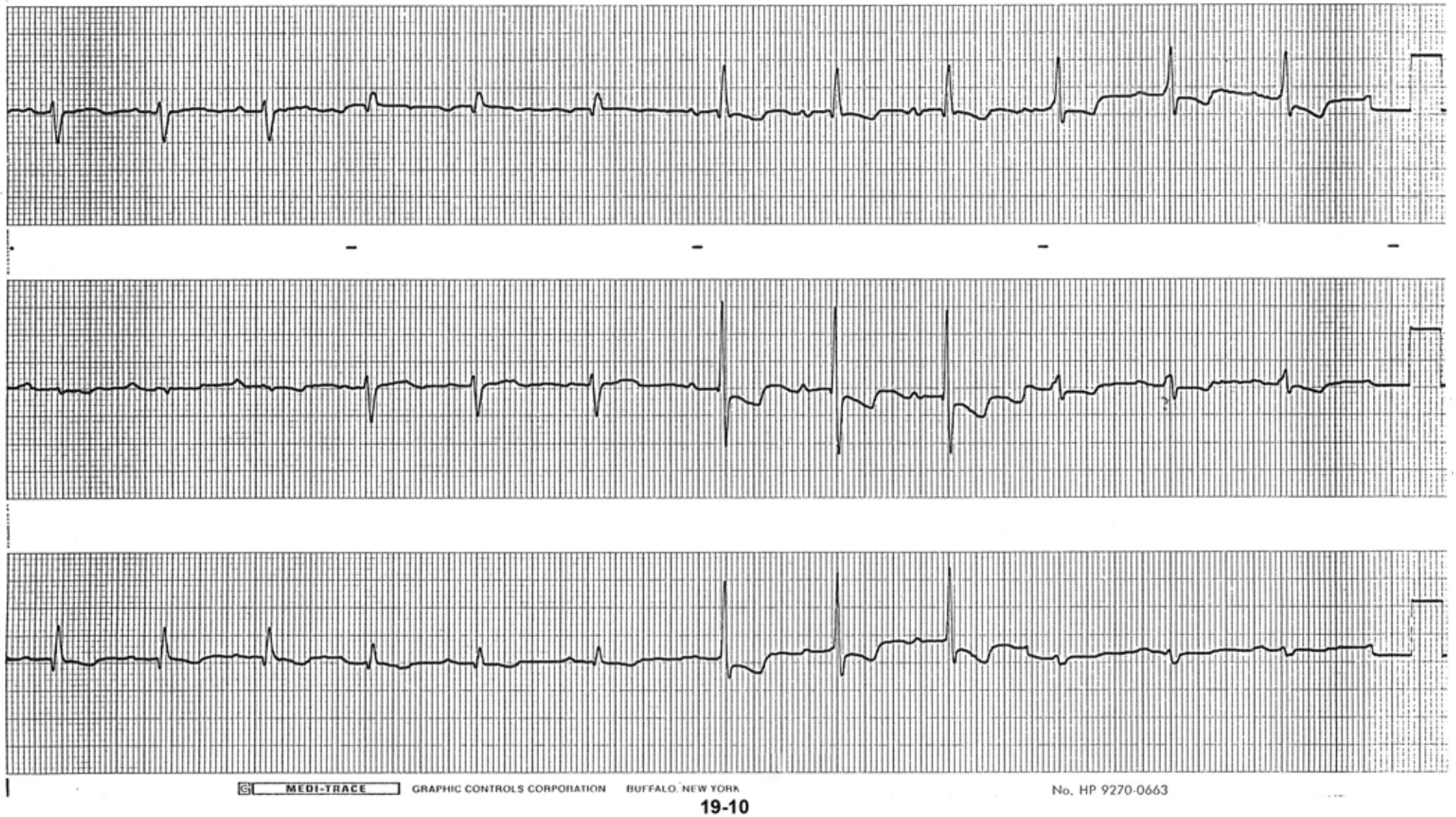
Dx: Preexcitation of every other beat initially simulating ventricular bigeminy, then every beat (three beats) at the end



19-9

19-9 Sinus tachycardia at a rate of 109/minute. Every other QRS is different looking, wider, and occurs slightly prematurely, suggesting atrial or junctional bigeminy with aberrant conduction or ventricular bigeminy. Or, the QRS alternates in its polarity, suggesting electrical alternans. However, the wider, different-looking QRS has a short P-R interval and a slurred upstroke typical of delta wave, indicating that it is a preexcited beat. This is an example of preexcitation affecting only every other beat. Preexcitation can be intermittent, affecting every other beat as in this case, several beats at a time, or several minutes at a time, etc.

Dx: Preexcitation of every other beat

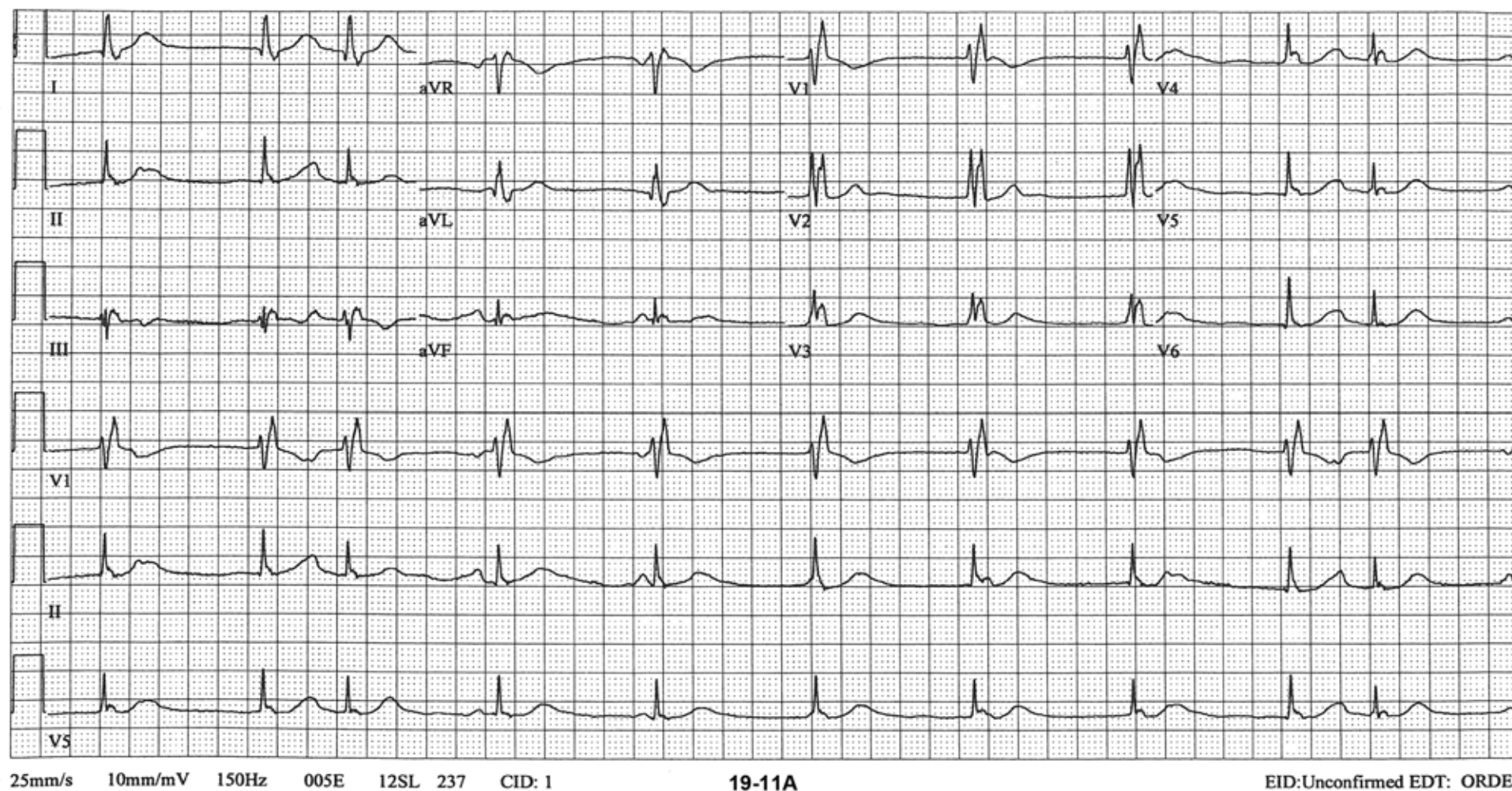


19-10 Normal sinus rhythm at a rate of 70/minute. RAD, tall R waves in the right precordial leads, S wave in V_6 , ST depression of a strain pattern in the right precordial leads are all diagnostic features of RVH.

- Dx:
1. NSR
 2. RVH

19-11 *Question:* In addition to RBBB, what are the rhythm and conduction problems? Choose one from the list below:

- A. Complete AV block, junctional escape rhythm, and two sinus beats
- B. Complete AV block, junctional escape rhythm, and two atrial premature beats
- C. Sinus bradycardia, junctional escape rhythm with AV dissociation, and three sinus (capture) beats

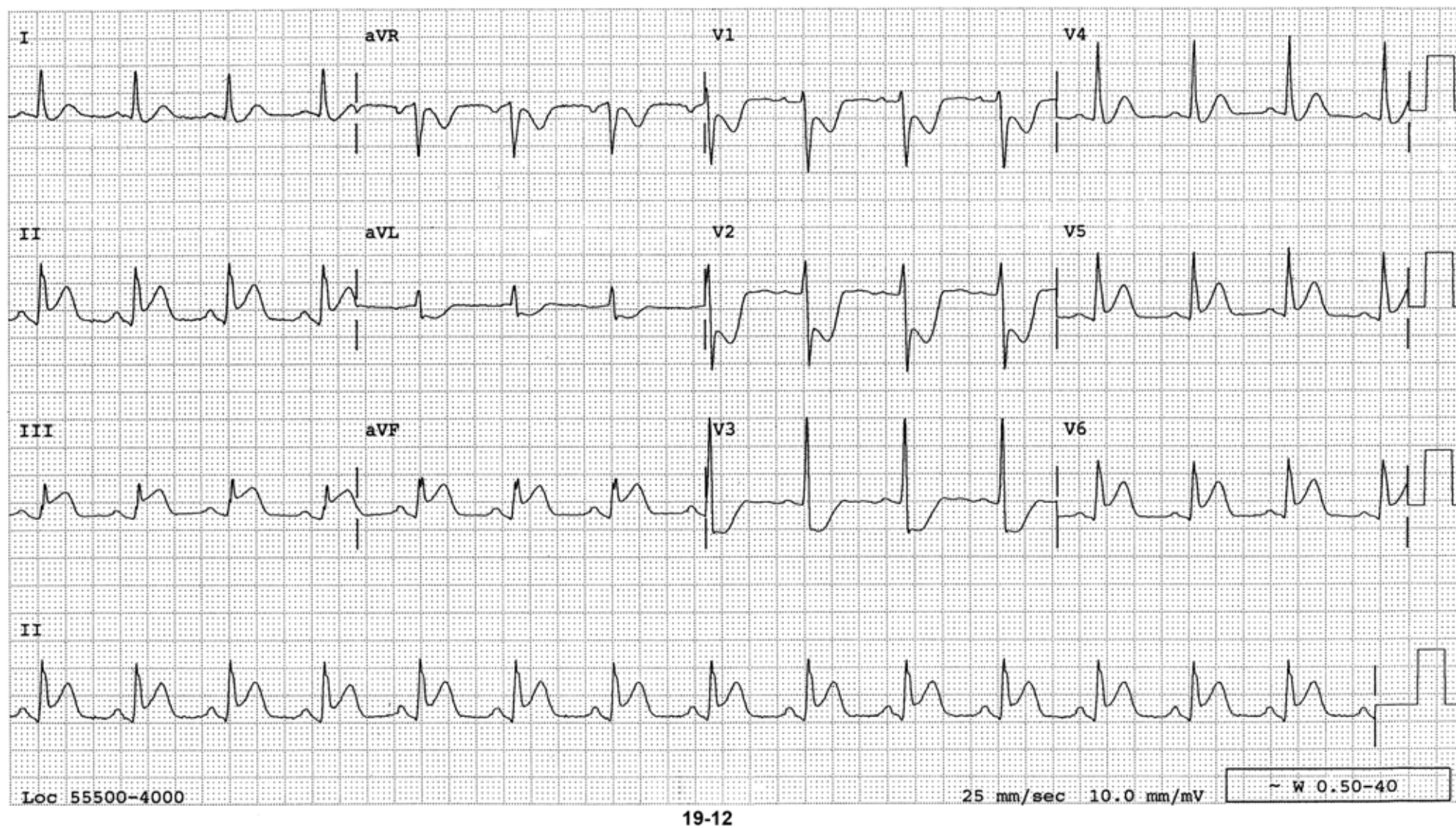




Answer: C. Sinus bradycardia, junctional escape rhythm with AV dissociation, and three sinus (capture) beats

Discussion: At a glance this appears to be a complicated case, but it is not. Let us take one beat at a time. First let us pick the beat that seems to have a normal PR relationship: P_3 . Let us assume that P_3 is conducted to R_4 . P_4 was going to be conducted to the ventricles, but the P-P interval is slightly longer than the junctional escape interval in this patient at the moment, and so the junctional beat (R_5) manifest (escapes). Because P_4 and R_5 are occurring close together during each other's physiologic refractory period, each interferes with the other's impulse propagation, resulting in AV dissociation. The same events are happening with P_5 - P_7 . R_3 and R_{10} occur earlier than other QRSs, raising a possibility of PAC or premature junctional complexes. However, P waves (\downarrow , P_2 and P_8) are in front of them, occur regularly, and these QRSs are conducted from the sinus impulse (capture beats). How do we know that R_4 is conducted from P_3 ? R_4 occurs with a shorter interval (1 second) than other QRSs, which occur with an interval of 1.08 second. The 80 millisecond difference is sufficient and conclusive evidence for R_4 not being a junctional beat and is conducted from P_3 . Thus, the primary problem is a simple, mild sinus bradycardia. Junctional escape, AV dissociation, and occasional capture (conducted) beats are all obligatory secondary manifestations. Capture beats are useful in that they prove that there is no AV block.

Sometimes the secondary manifestations may make the tracing look more complicated and troublesome when the primary problem may be as simple as mild sinus bradycardia.

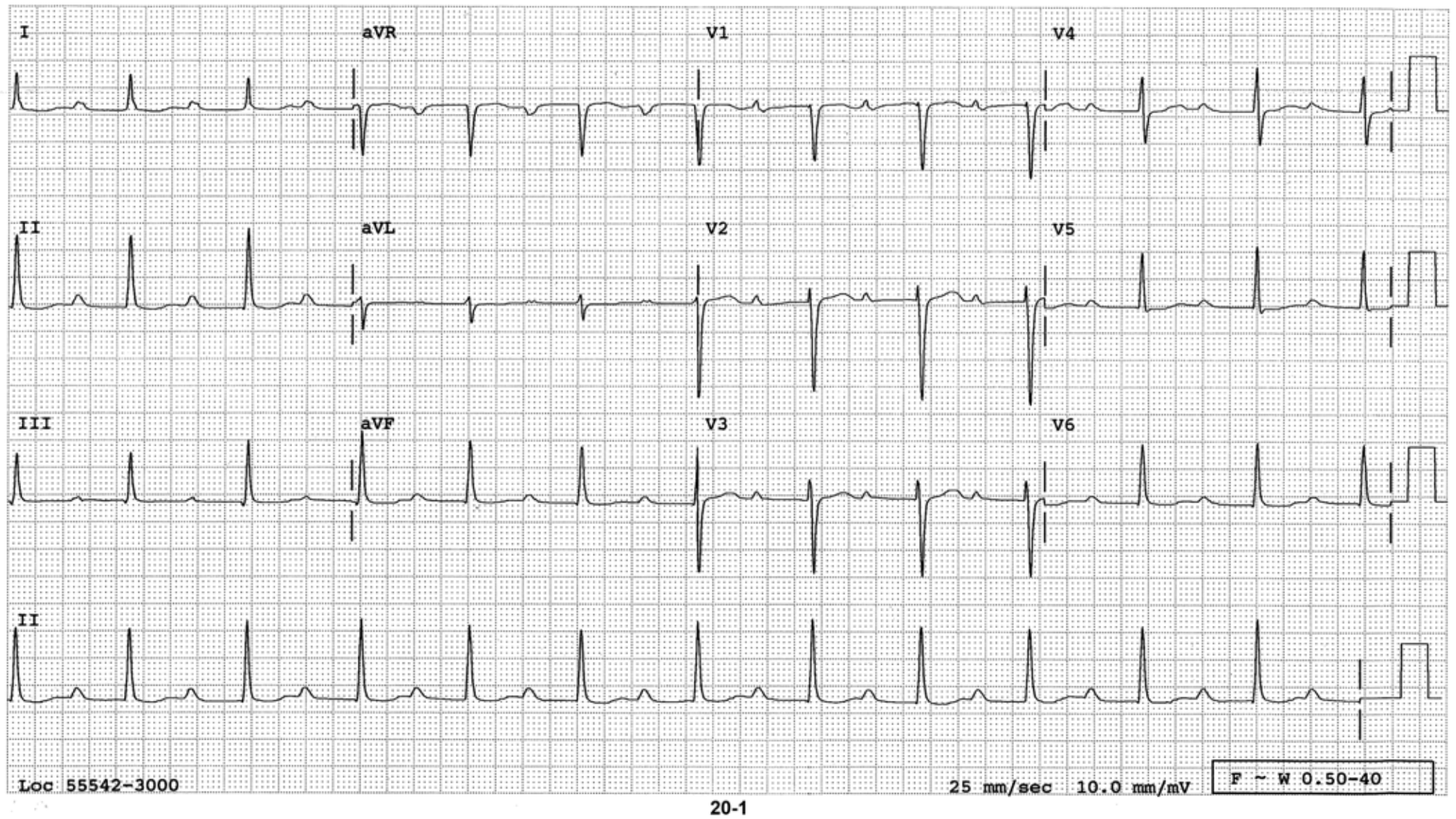


19-12

19-12 Normal sinus rhythm at 86/minute ST elevation in inferior leads and V_5 - V_6 combined with horizontal ST depression in V_1 - V_3 is diagnostic of acute inferoposterolateral MI. ST is reciprocally depressed only in aVL, not in lead I, indicating RV is not involved and the culprit lesion must be in circumflex coronary artery or RCA not proximal.

- Dx:*
1. NSR
 2. Acute inferoposterolateral MI

SECTION 20



20-1 A regular rhythm with narrow QRSs at a rate of 72/minute. The P-R interval is markedly prolonged to 360 milliseconds. Yet, no P waves are blocked indicating 1° AV block.

- Dx: 1. NSR
2. 1° AV block

20-2 Question: This is simultaneous recordings of the ECG and arterial pressure tracings. Why does the systolic pressure alternate?



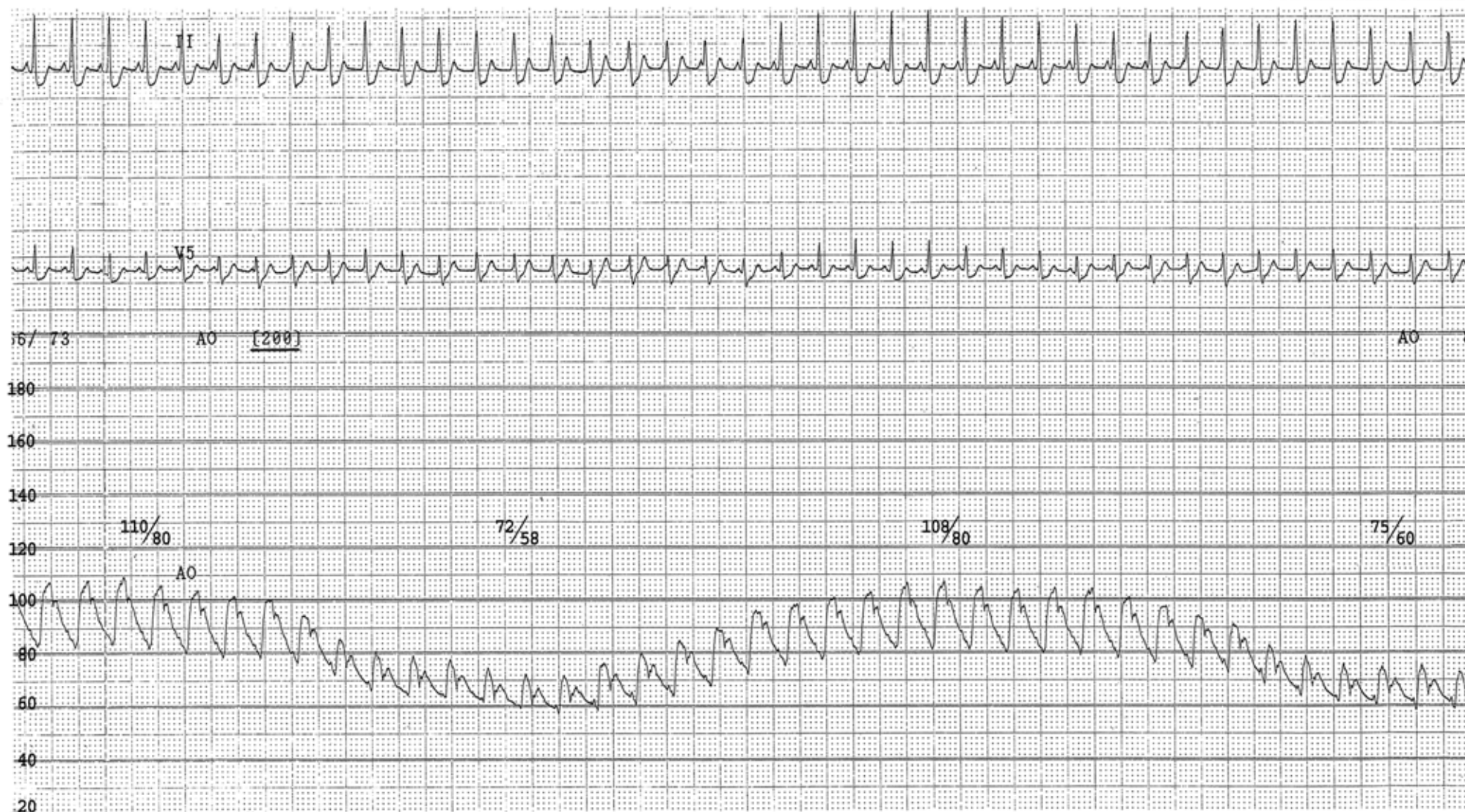
20-2

Answer: Having or not having a P wave (“atrial kick”) in front of the QRS complex, a manifestation of the Frank-Starling’s law

Discussion: This patient is paced in the ventricle for complete (3°) AV block. It happens that every other QRS complex has a P wave in front of it. The sinus rate is about twice the ventricular rate and other P waves are lost in the T waves or QRS complexes. The beat with a P wave in front of it has the atrial contribution to the stroke volume, resulting in a higher systolic pressure than the beat with no P wave in front of it; a manifestation of the Frank-Starling’s law, which states when a muscle fiber is stretched, it will contract more forcefully.

20-3 Question: This is simultaneous recordings of the cardiac rhythm and arterial pressure (BP). What is the reason for the gradual fluctuations of the arterial pressure? Choose one from the list below:

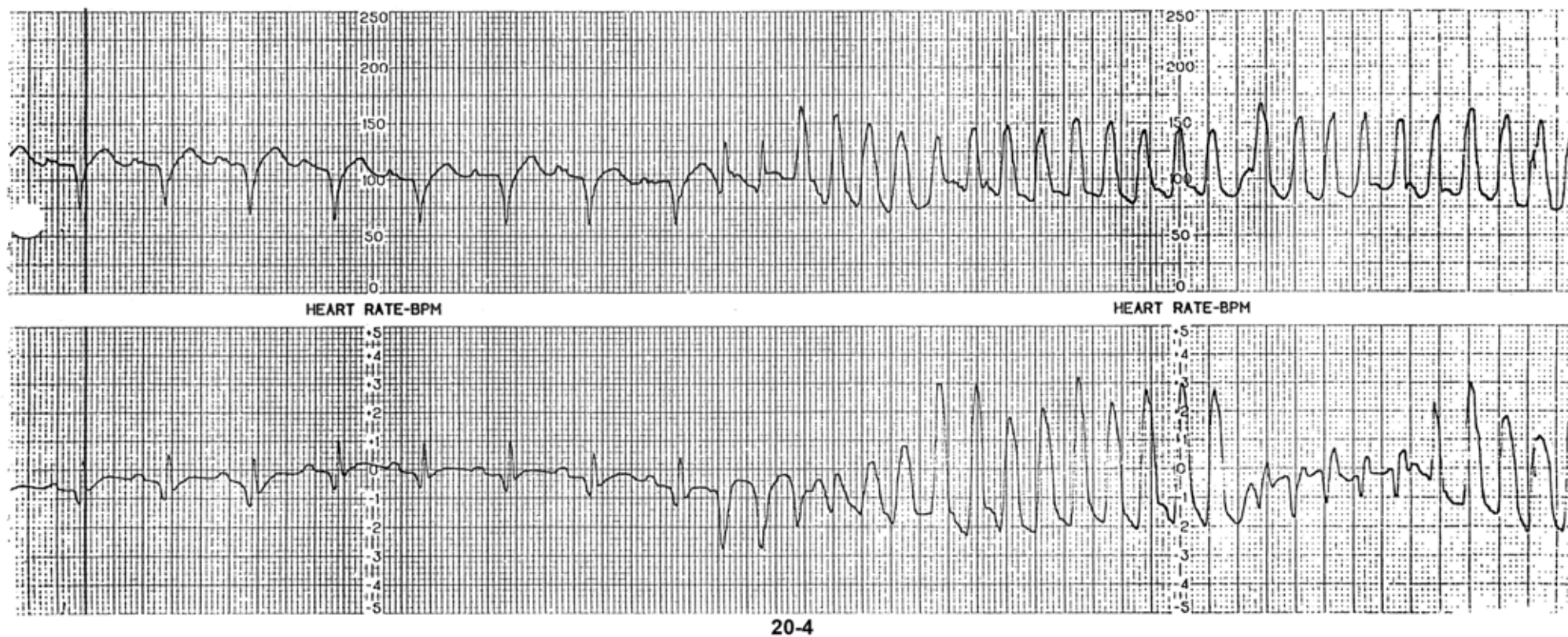
- A. Paradoxical pulse due to either cardiac tamponade or marked swing in the intrathoracic pressure
- B. Having or not having a P wave (an atrial kick) before the QRS complex
- C. Normal respiratory response



20-3

Answer: B. Having or not having a P wave (an atrial kick) before the QRS complex

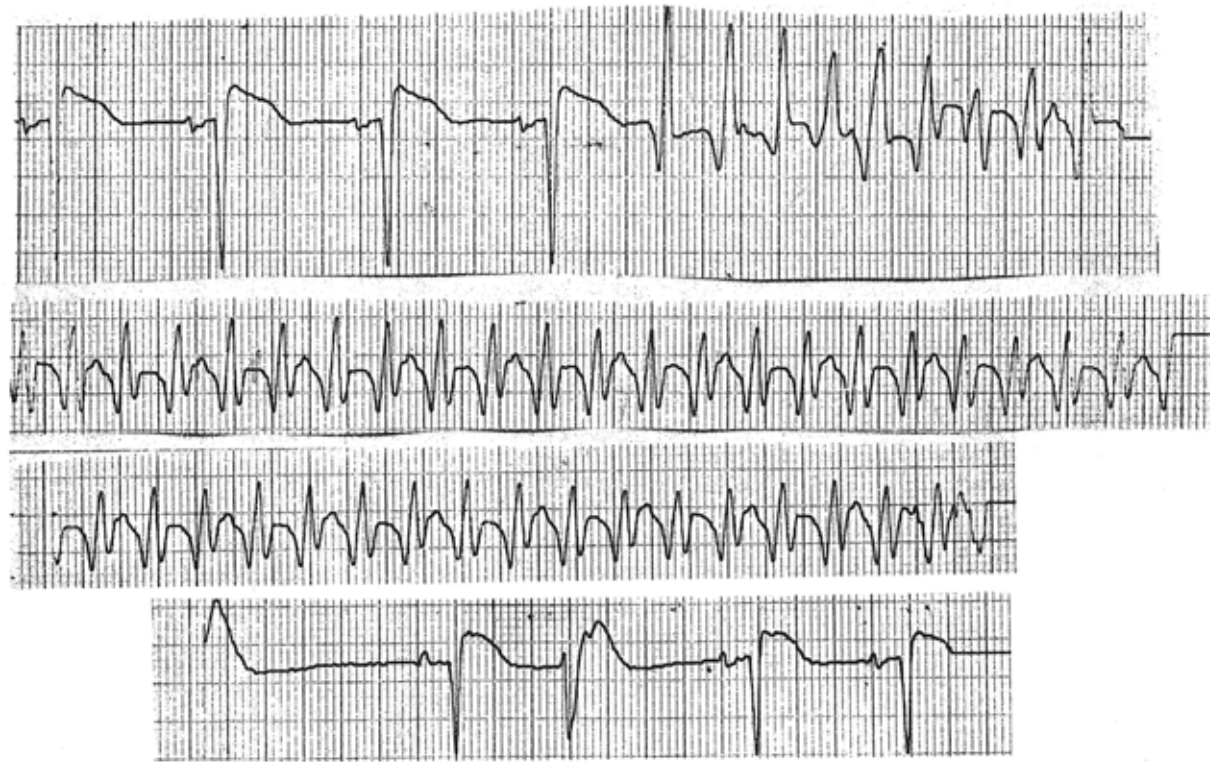
Discussion: This patient has accelerated junctional rhythm with AV dissociation. For several beats, the P wave occurs before the QRS complex resulting in physiologic AV sequential relationship while at other times the P wave occurs within the QRS complex. Thus, the patient gains and then loses the atrial “kick”, causing marked fluctuation in the stroke volume. This is manifested as fluctuating blood pressure, an expression of the Frank-Starling’s law. It is not (A) or (C) because the cycle frequency is about 10/minute, which is too slow for respiration. Besides, 30 mm Hg swing in blood pressure is not a normal respiratory response.



20-4

20-4 The rhythm strip reveals NSR initially followed by a wide QRS tachycardia with a changing QRS morphology at a rate of about 250/minute which is good for polymorphic VT. Since the Q-T interval is not prolonged during sinus rhythm, this rhythm will not be called *Torsade de Pointes*.

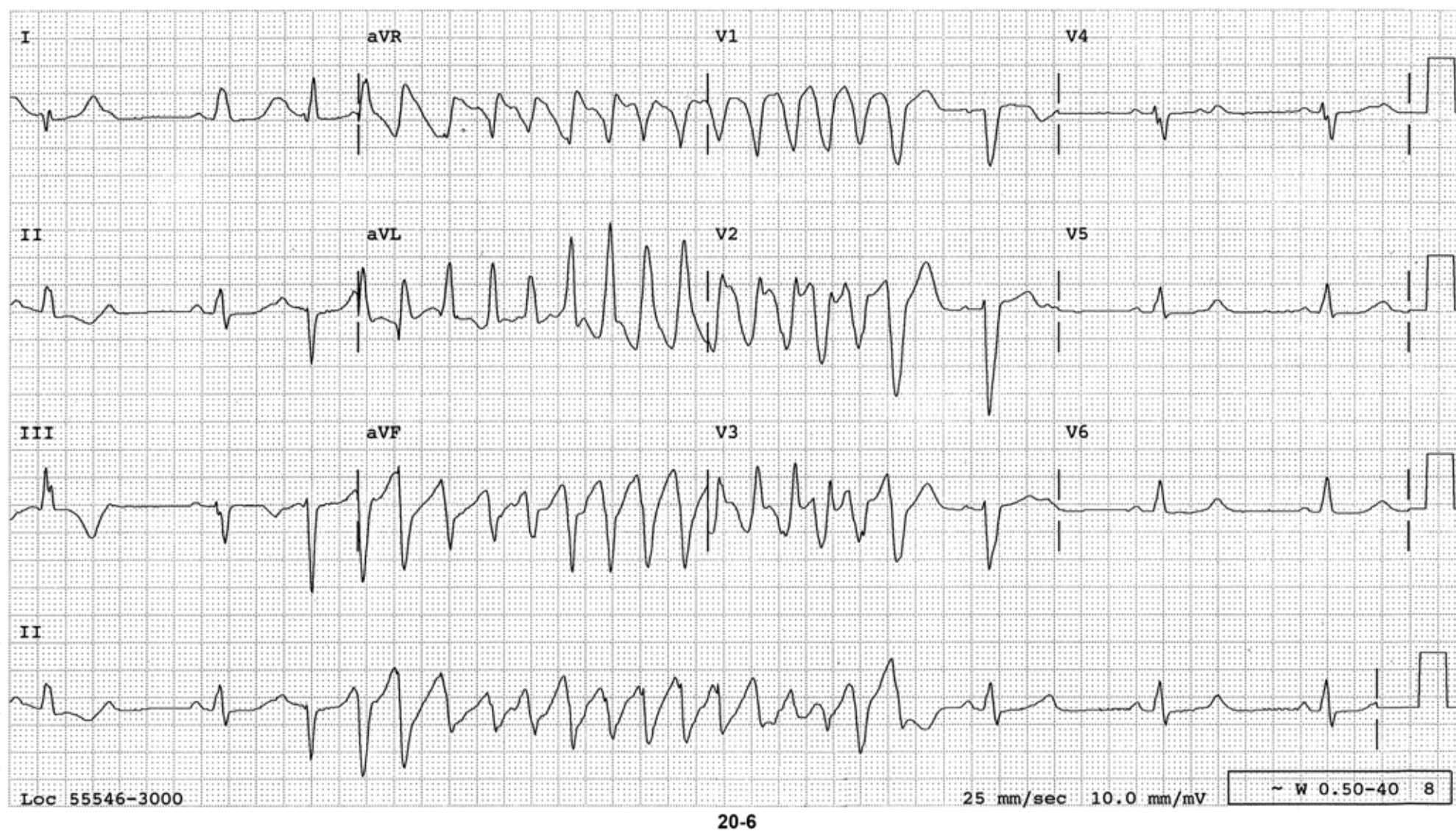
Dx: NSR followed by polymorphic VT



20-5

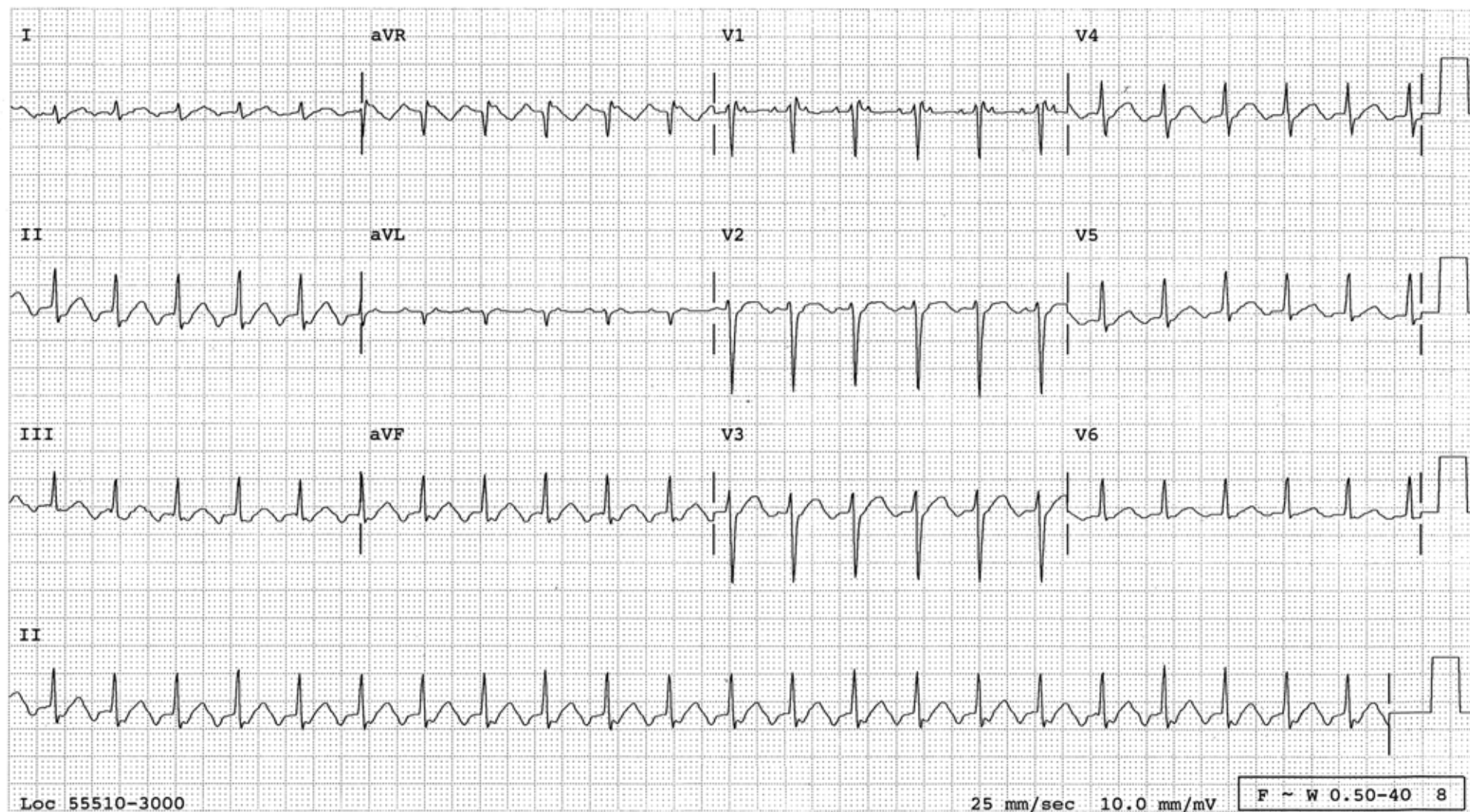
20-5 A regular sinus rhythm is interrupted by a wide QRS tachycardia which spontaneously converts to sinus rhythm again. The first complex of this wide QRS tachycardia is not preceded by a P wave proving that this rhythm originates from the ventricle. Initially, this VT is polymorphic which settles down to monomorphic. An interesting feature of this tracing is in the middle tracings. There is a bump on every other T wave while the other T wave is smooth. This is an example of VT with 2:1 VA conduction resulting in a retrograde P wave for every other complex.

Dx: Normal sinus rhythm followed by a run of VT with 2:1 VA conduction which converts back to sinus rhythm with a PVC



20-6 Sinus rhythm at a rate of 50/minute. The Q-T interval is very long measuring about 600 milliseconds (see V₅ and V₆). In the middle of the tracing there is a polymorphic VT. Since this occurs in the setting of a long Q-T interval, this will be called *Torsade de Pointes*. Depending upon the duration of this rhythm, this may cause either syncope or even sudden death. The treatment of choice is magnesium given intravenously. The cause of long Q-T interval should be sought for and if it is medication-induced, the offending agent should be stopped (the offending agent in this patient was sotalol).

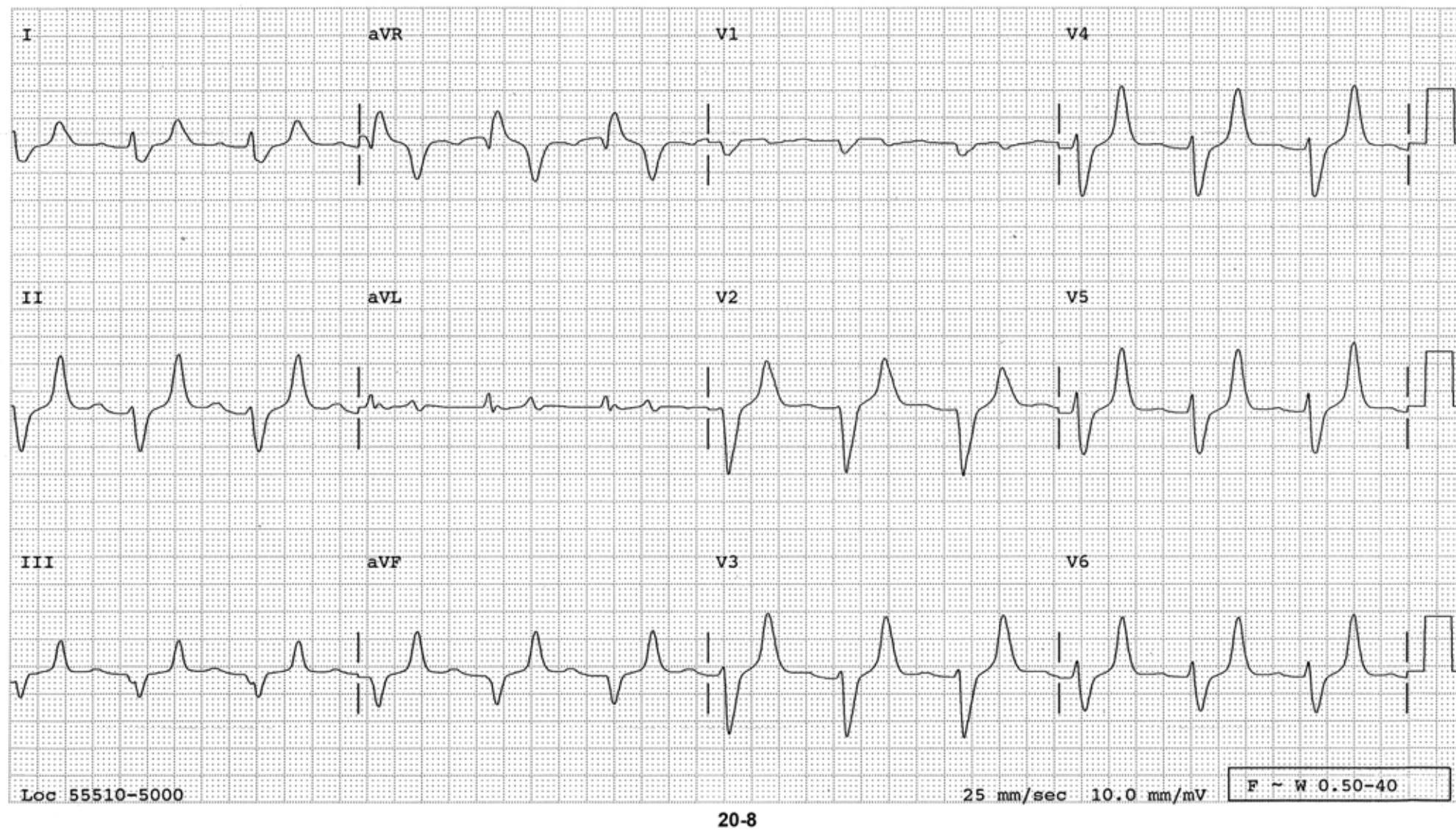
- Dx:
1. NSR
 2. Long Q-T interval
 3. *Torsade de Pointes*



20-7

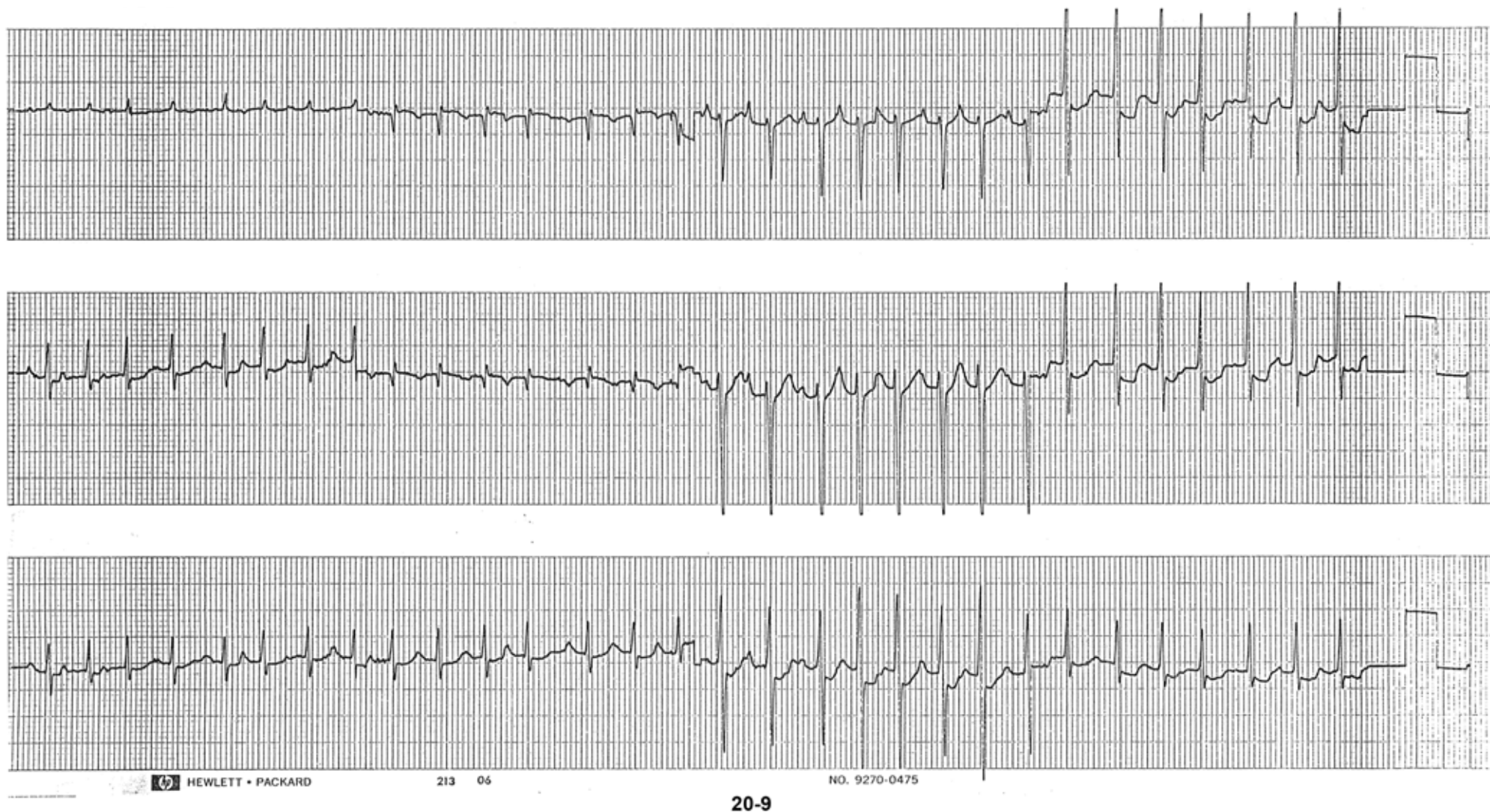
20-7 A narrow QRS tachycardia at a rate of 135/minute. The sawtooth pattern of flutter waves is recognizable in the inferior leads. If there is any doubt, lead V₁ which reveals two atrial activities for each QRS is very helpful.

Dx: Atrial flutter with 2:1 AV conduction



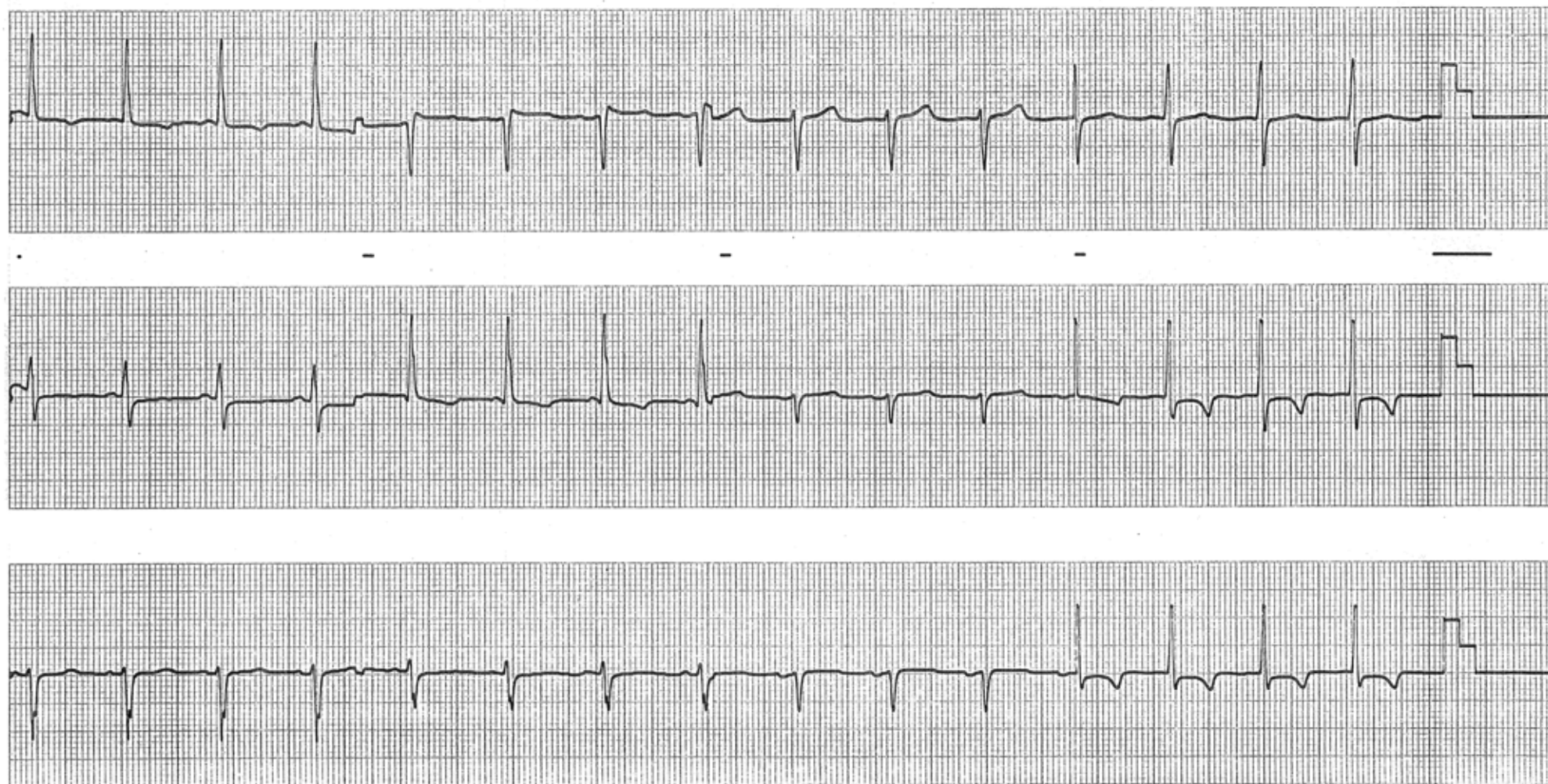
20-8 Wide QRSs occur regularly. P waves can be recognized in inferior leads with a long P-R interval. In other leads, the P waves are relatively flat. The T waves are tall, pointed and narrow in many leads. All of these features are diagnostic of hyperkalemia.

- Dx:
1. Sinus rhythm
 2. 1° AV block
 3. Findings consistent with hyperkalemia



20-9 Narrow QRS with irregularly irregular rhythm at a rate of 170/minute is present. P waves are not clearly recognizable suggesting atrial fibrillation. However, in V_1 there is a well-organized P wave in front of each QRS which occurs irregularly with a changing morphology and it is diagnostic of MAT. ST-T changes are present in the left precordial leads. As in this case, sometimes only one lead may show the diagnostic findings convincingly.

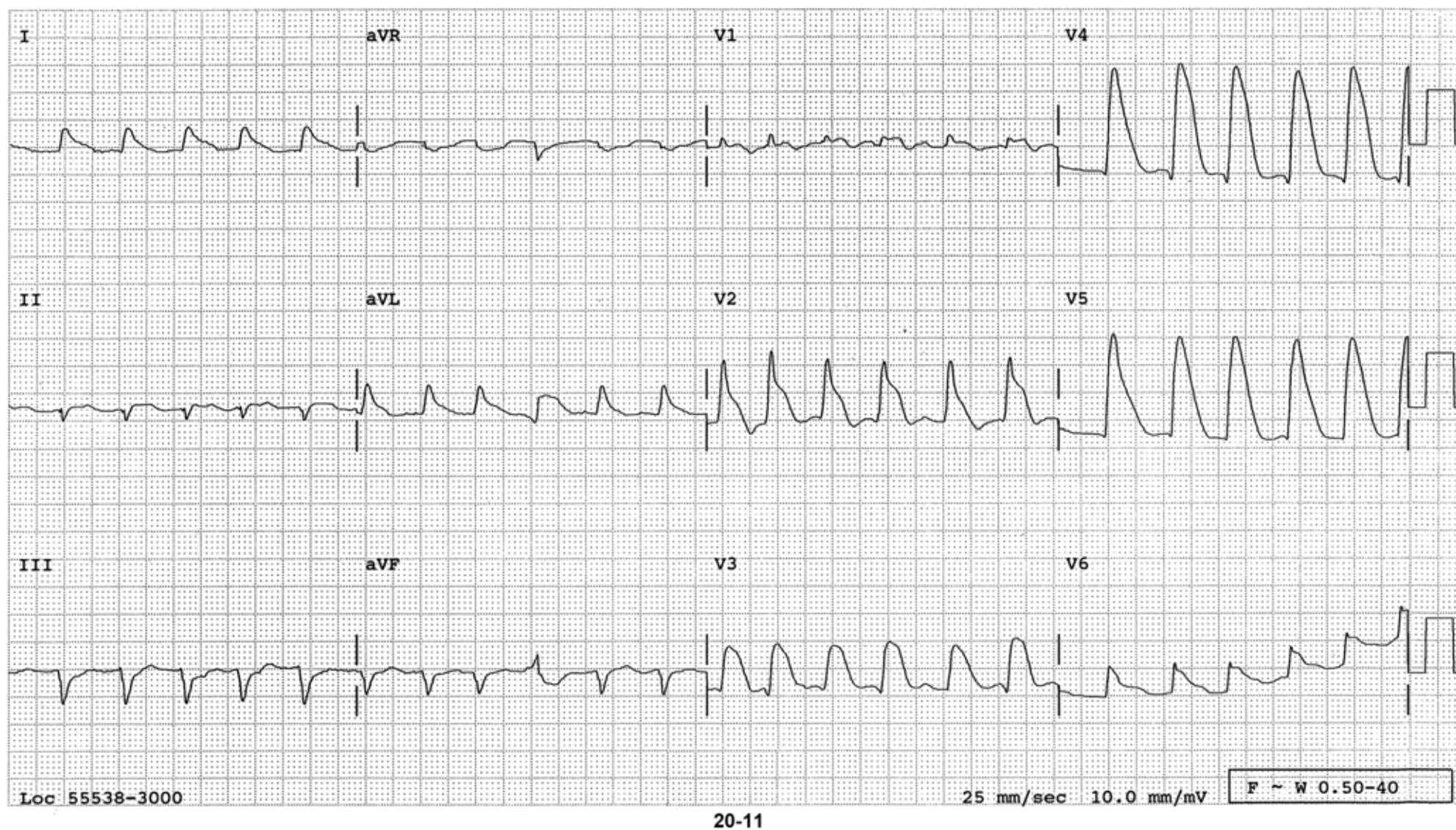
- Dx: 1. MAT
2. ST-T changes



20-10

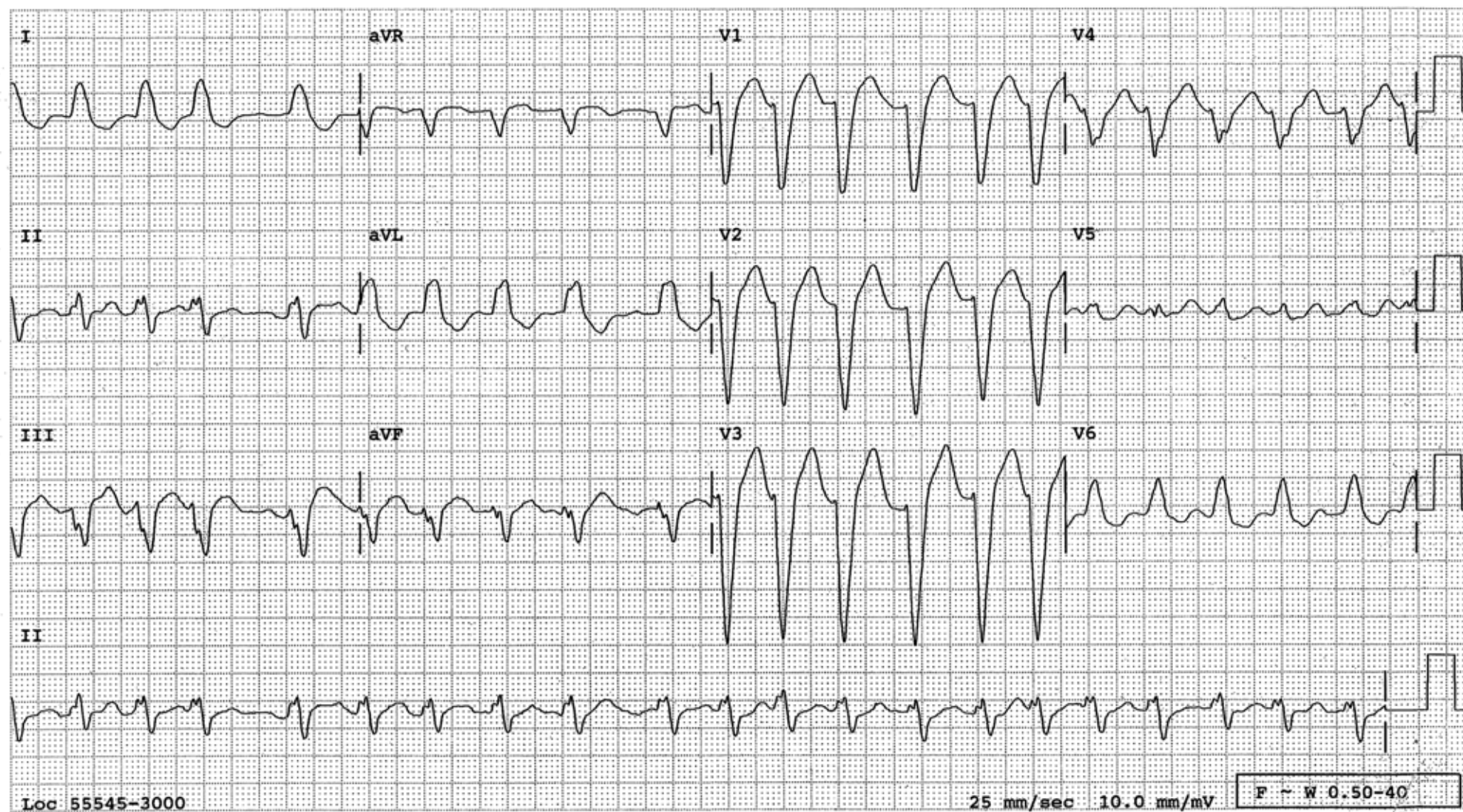
20-10 Normal sinus rhythm at a rate of 89/minute. Voltage criteria and ST-T changes for LVH are present (note that the precordial leads are half-standard). The R waves are regressing rather than progressing in the right precordial leads. Actually, V_1 and V_3 leads are reversed judging from the fact that the P wave in V_3 is mostly negative which belongs to V_1 and not to V_3 . Another example of the usefulness of the P wave morphology:

- Dx:*
1. Sinus rhythm
 2. LVH
 3. Reversed V_1 V_3 leads



20-11 Irregularly irregular rhythm at a rate of 138/minute with no definite P waves indicating atrial fibrillation. The ST-segment is markedly elevated which is better appreciated in V_2 and is diagnostic of STEMI. This marked ST elevation simulates widened QRS in other leads such as V_3 - V_5 . There is an abnormal left axis deviation consistent with LAFB.

- Dx:*
1. Atrial fibrillation
 2. LAFB
 3. Acute, extensive anterior infarct



20-12

20-12 Irregularly irregular rhythm at a rate of 129/minute with no definite P waves indicating atrial fibrillation. The QRS is wide and has a typical LBBB pattern. If the rate is faster, one can see how this tracing could easily be mistaken for a run of VT.

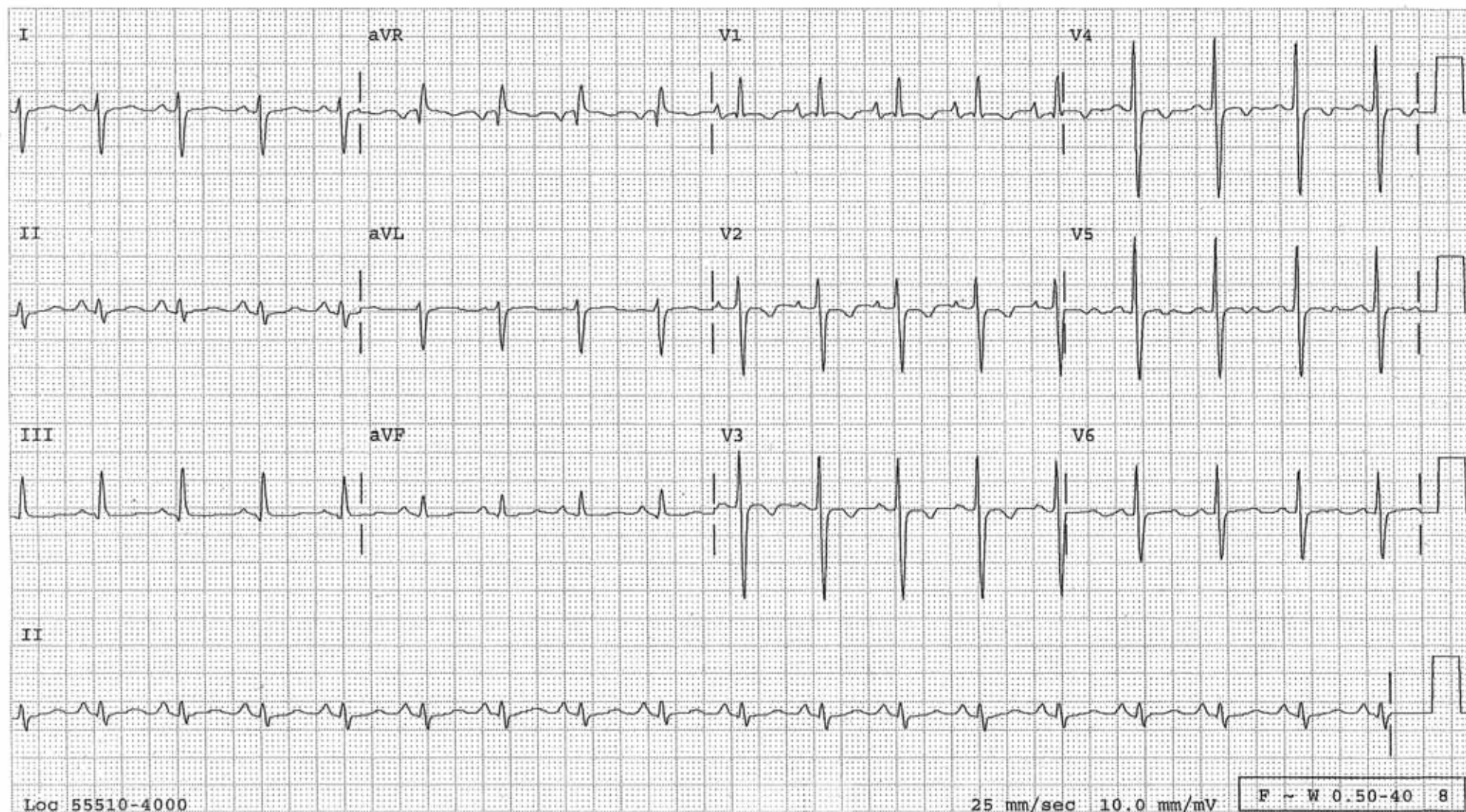
- Dx: 1. Atrial fibrillation with a ventricular response of 129/minute
2. LBBB

SECTION 21

INTRODUCTION TO THE QUESTIONS 21-1 THROUGH 21-12

Differential diagnosis of tall R waves in the right precordial leads:

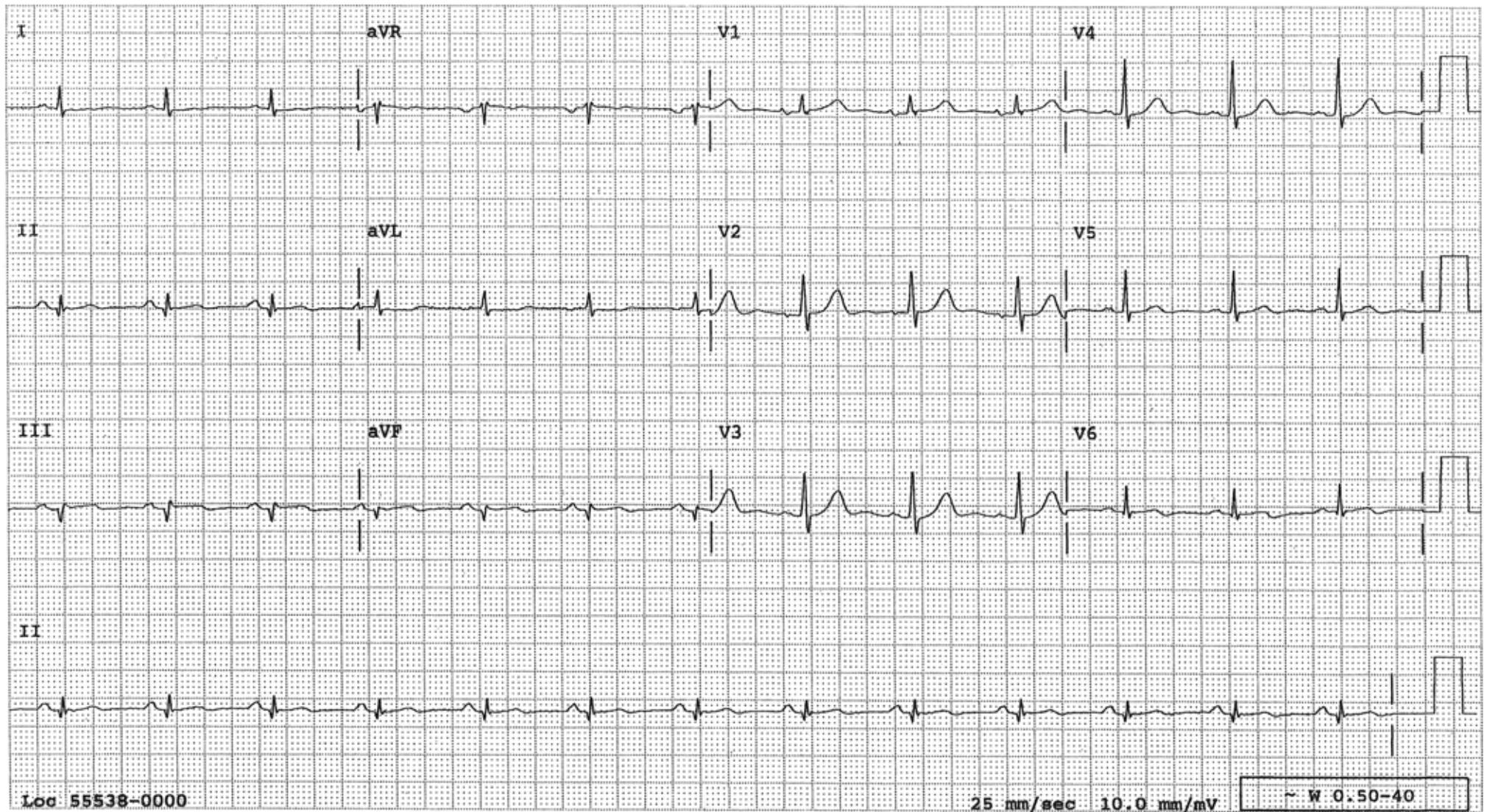
- A. *RVH*: RAD and deep S waves in V_6 and ST-T changes (strain pattern) in V_1 - V_3
- B. *Posterior infarction*: Often supporting evidence is present in the inferior or lateral leads since it occurs often as inferoposterior or posterolateral infarction.
Acute: Horizontal ST depression in the right precordial leads which is an injury pattern ST elevation of posterior wall registered reciprocally.
Old: Just tall R waves with no ST-T changes
- C. *Some cases of WPW syndrome*: Short P-R interval and typical delta waves are present in some or all leads
- D. *Reversed precordial leads*: The lead with the most biphasic (initially positive, then negative) P wave belongs to V_1
- E. RBBB with or without anteroseptal MI



21-1

21-1 Dx: RVH

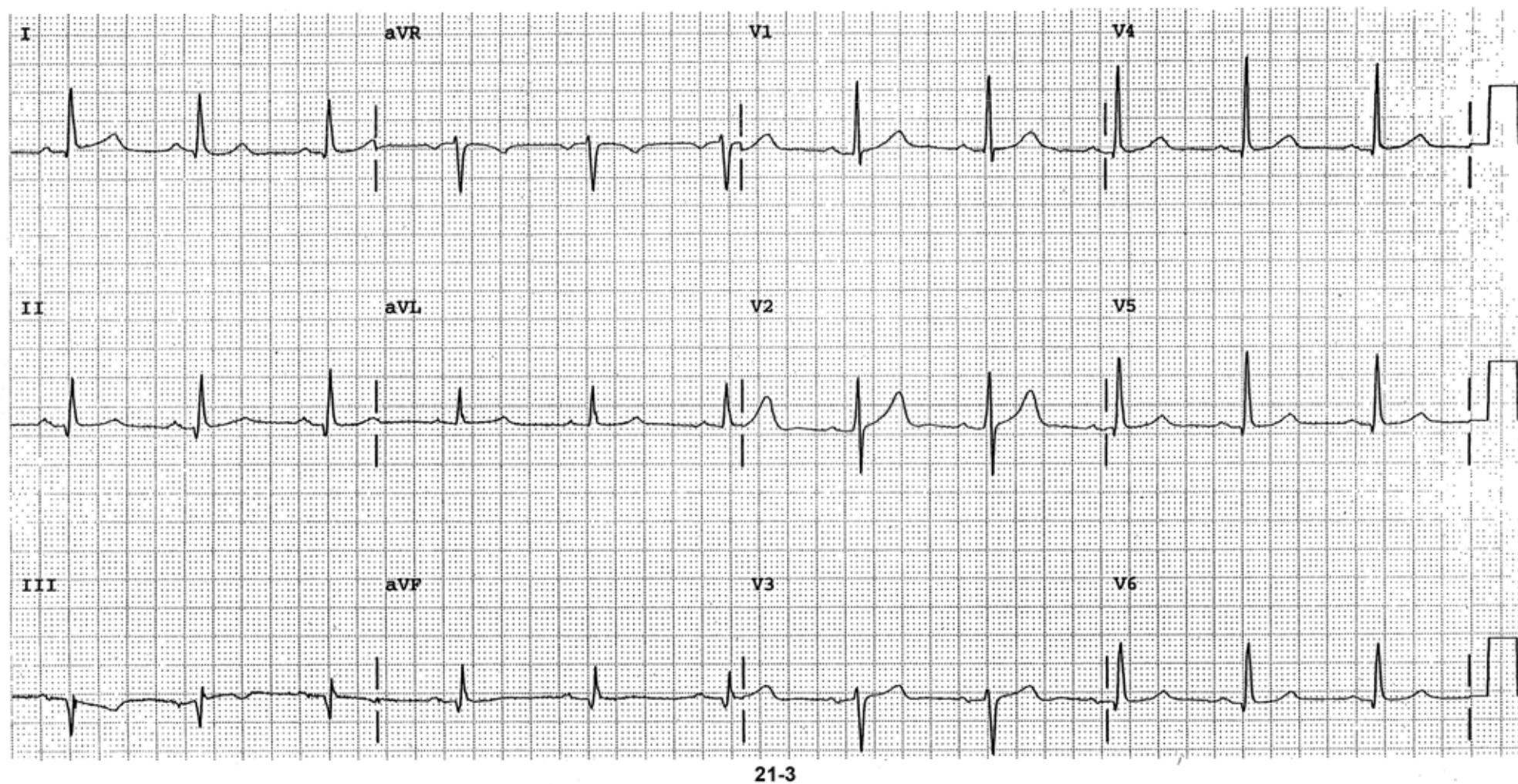
Salient features: Right axis deviation, tall R wave in V_1 , T wave inversion in the right precordial leads, S waves in the left precordial leads are all diagnostic features of RVH.



21-2

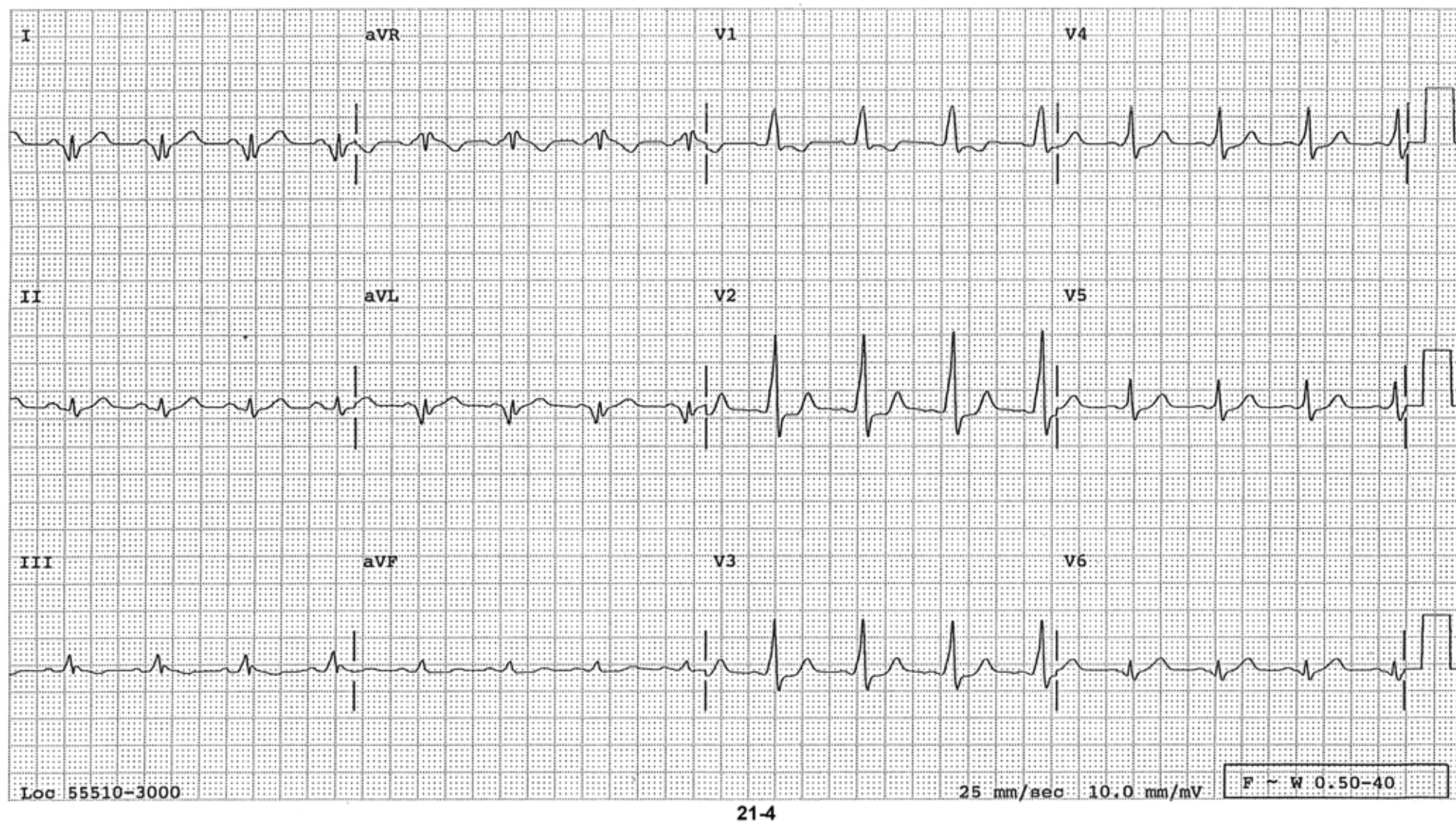
21-2 Dx: Inferoposterior MI

Clues: Q waves in the inferior leads are diagnostic of inferior MI, which involves the posterior wall as the cause of tall R waves in the right precordial leads. It is not RVH because there is no RAD and there are no prominent S waves in the left precordial leads. It is not WPW syndrome because the P-R interval is not less than 120 milliseconds and there are no delta waves in any leads. It is not reversed leads since the P wave is most biphasic in V_1 which belongs where it is and is entirely positive in V_3 which is appropriate.



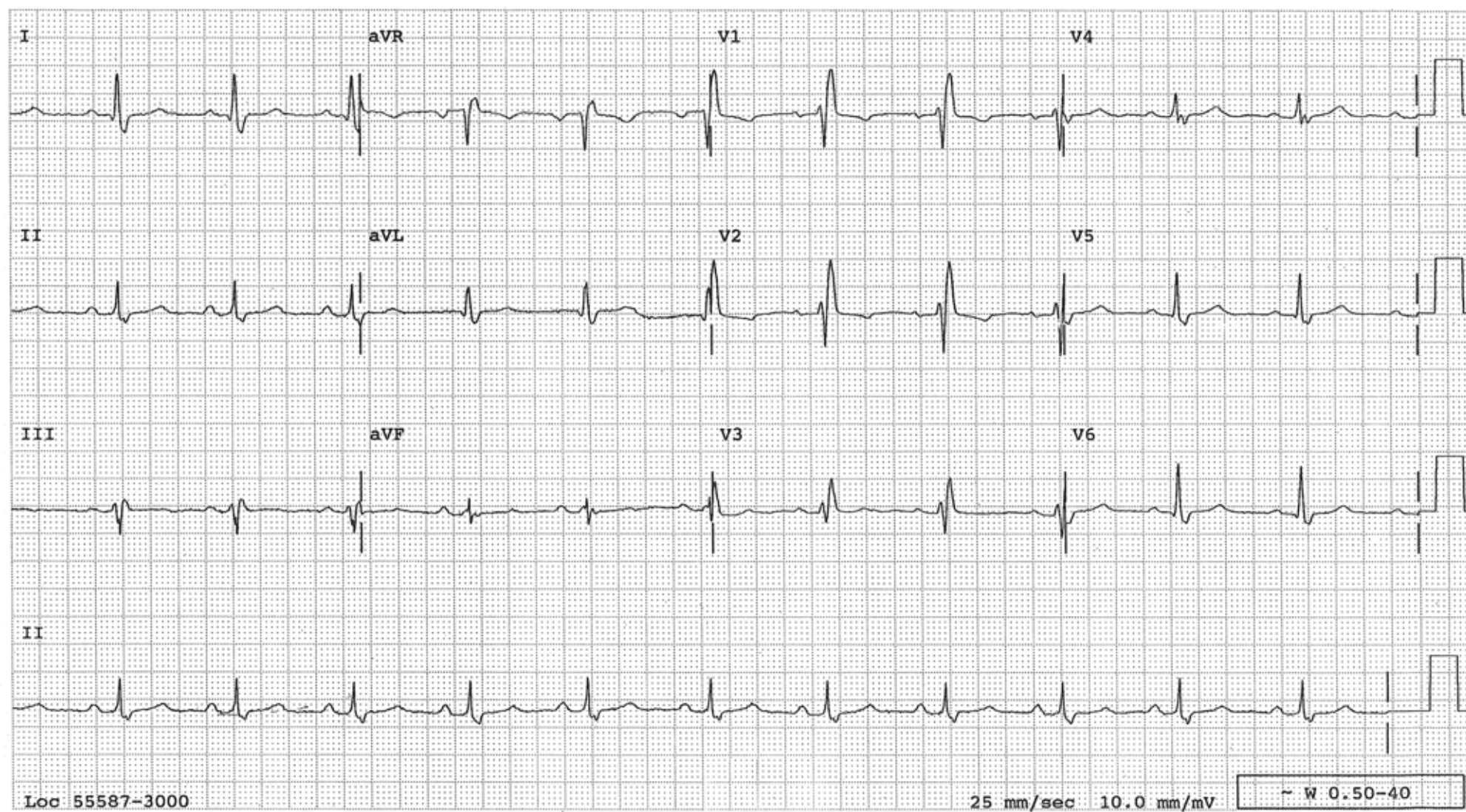
21-3 Dx: Inferior MI and reversed V_1 and V_3 leads

Clues: Inferior MI is obvious. The P wave is biphasic in V_3 and entirely positive in V_1 proving that these leads are reversed.



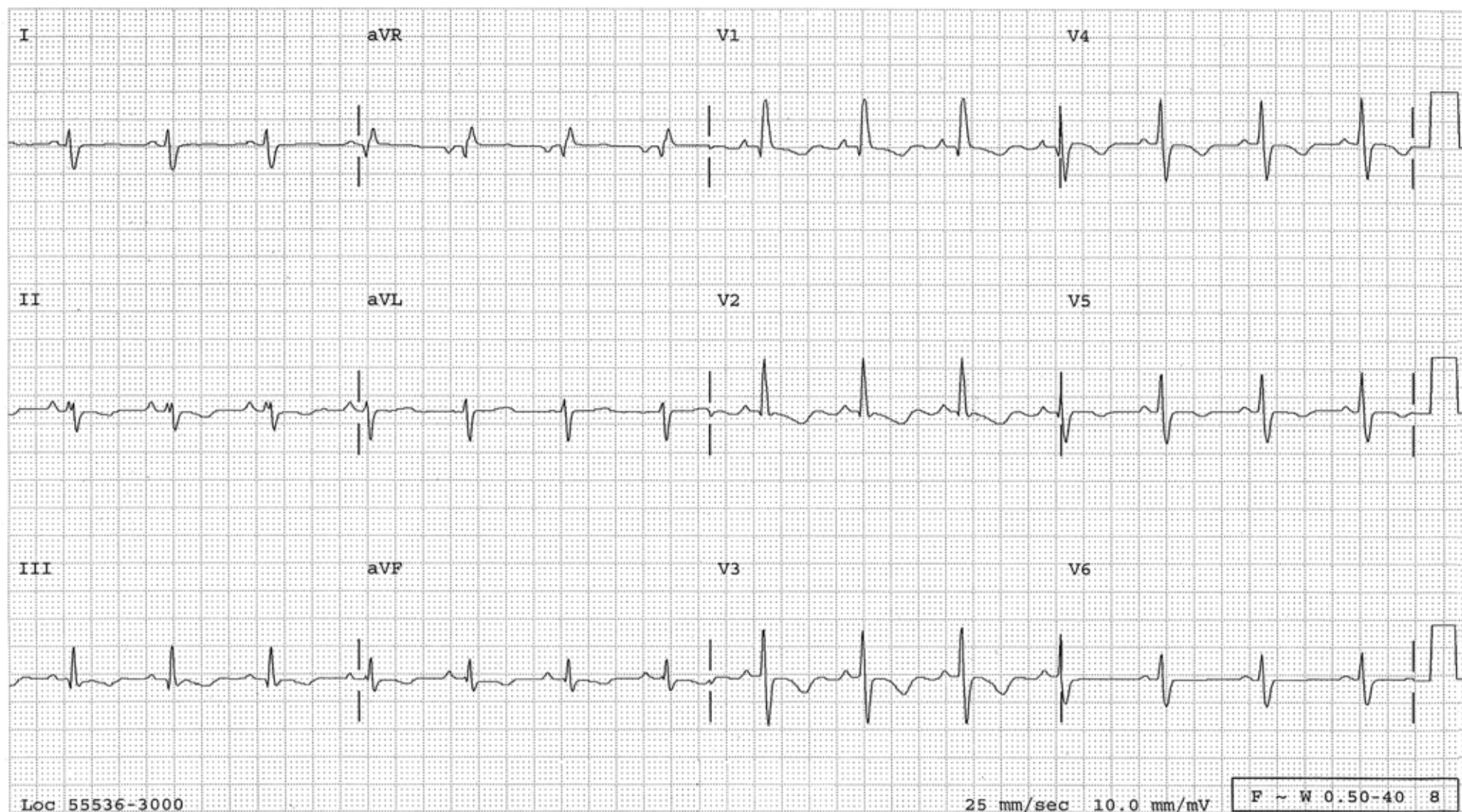
21-4 Dx: WPW syndrome

Clues: At first glance, it appears to be posterolateral MI with pathologic “Q waves” in leads I and aVL. However, the P-R interval is short in the precordial leads, and slurred upstroke of typical delta wave is present. This delta wave is directed from the patient’s left to right, registering as a negative delta wave in leads I and aVL, simulating lateral MI. In lead II, the P-R interval is normal and no delta wave is seen because the delta wave is isoelectric in that lead.



21-5

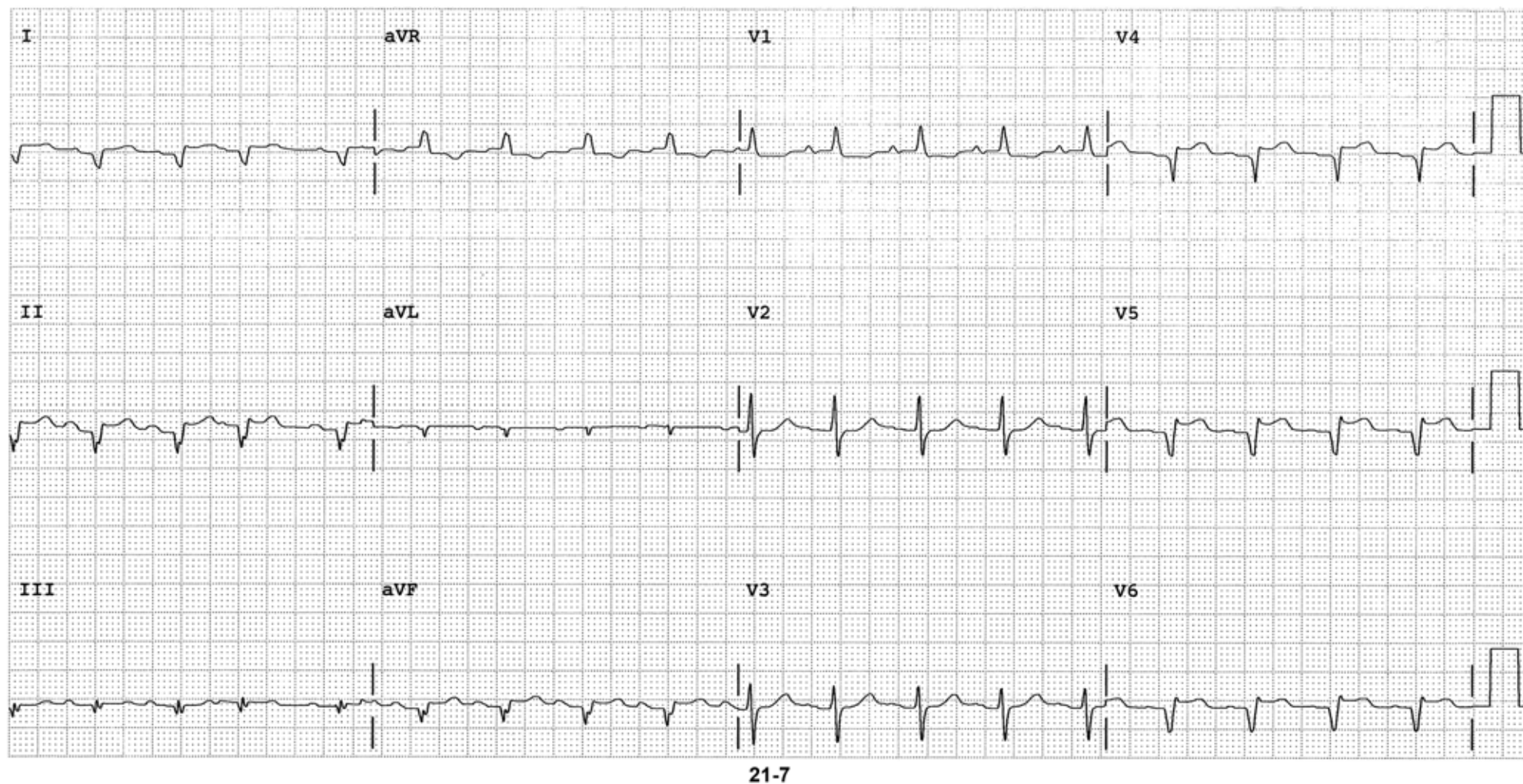
21-5 An example of typical RBBB.



21-6

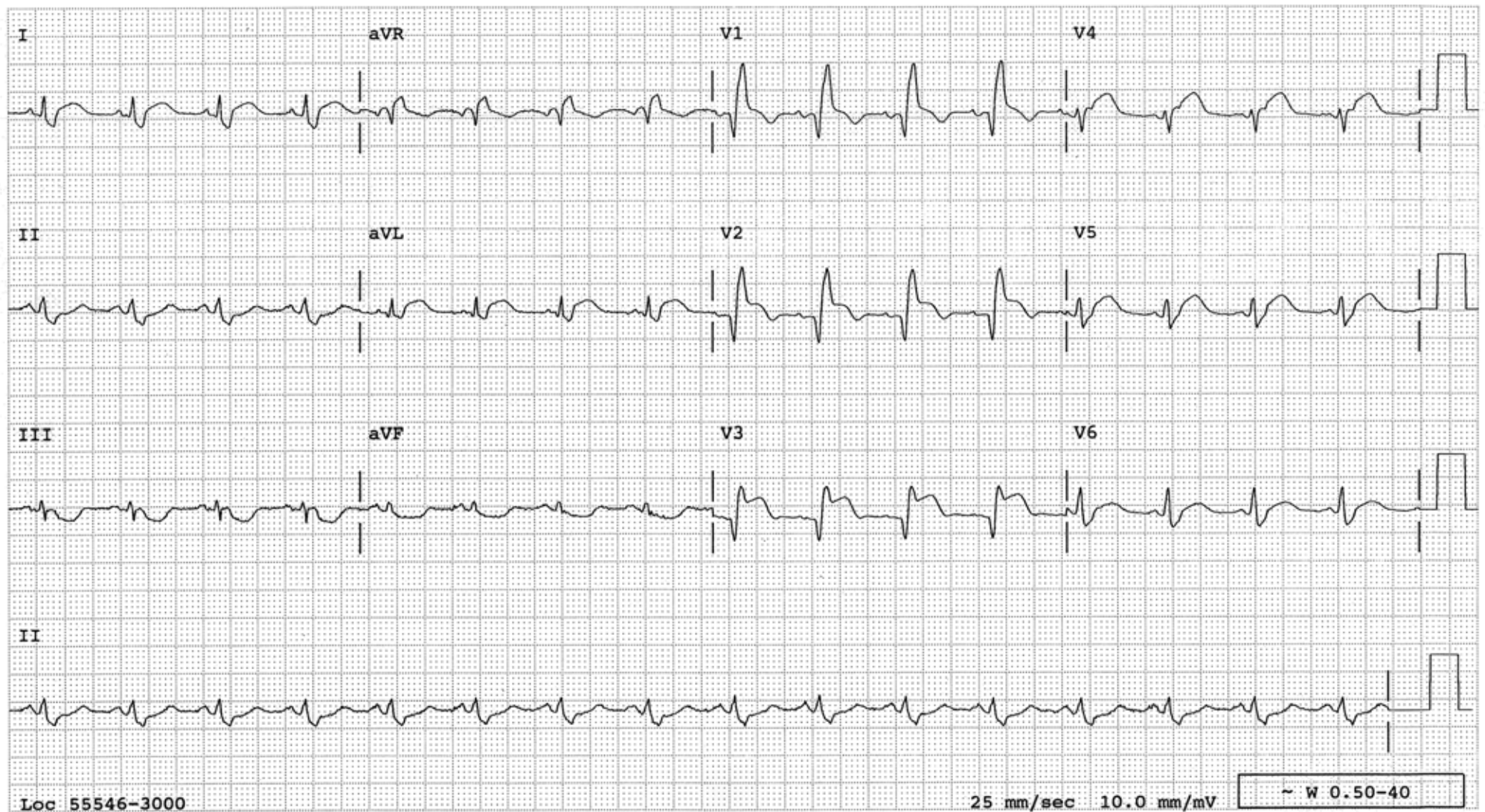
21-6 Dx: RVH

Clues: Right axis deviation, deep S wave in V_6 , tall R wave in V_1 , and T wave inversion predominantly in the right precordial leads, are all diagnostic features of RVH.



21-7 Dx: Inferoposterolateral MI

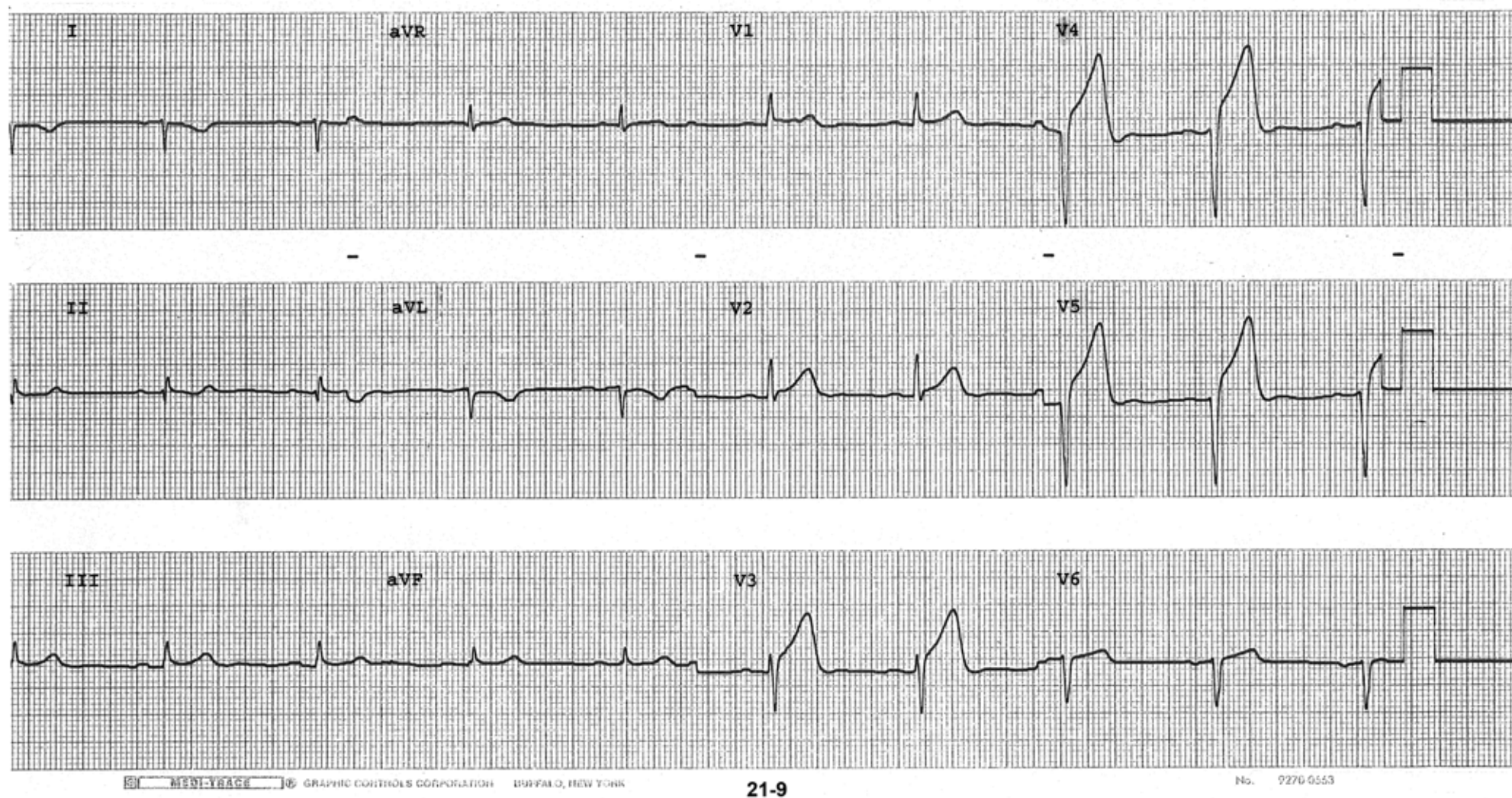
Clues: The P wave is positive in lead I, dispelling the notion of reversed arm leads or dextrocardia. The P waves are positive in the left precordial leads and the QRS morphology is similar in leads I, aVL and V_6 , indicating the precordial leads are regular left-sided. The P-R interval is not short in any of the 12 leads and there are no delta waves. The RAD in RVH has an rS pattern in lead I, not a QS or Qr pattern as in this patient. Also in RVH, there is an rS or Rs pattern in V_6 , not a Qr pattern as in this patient.



21-8

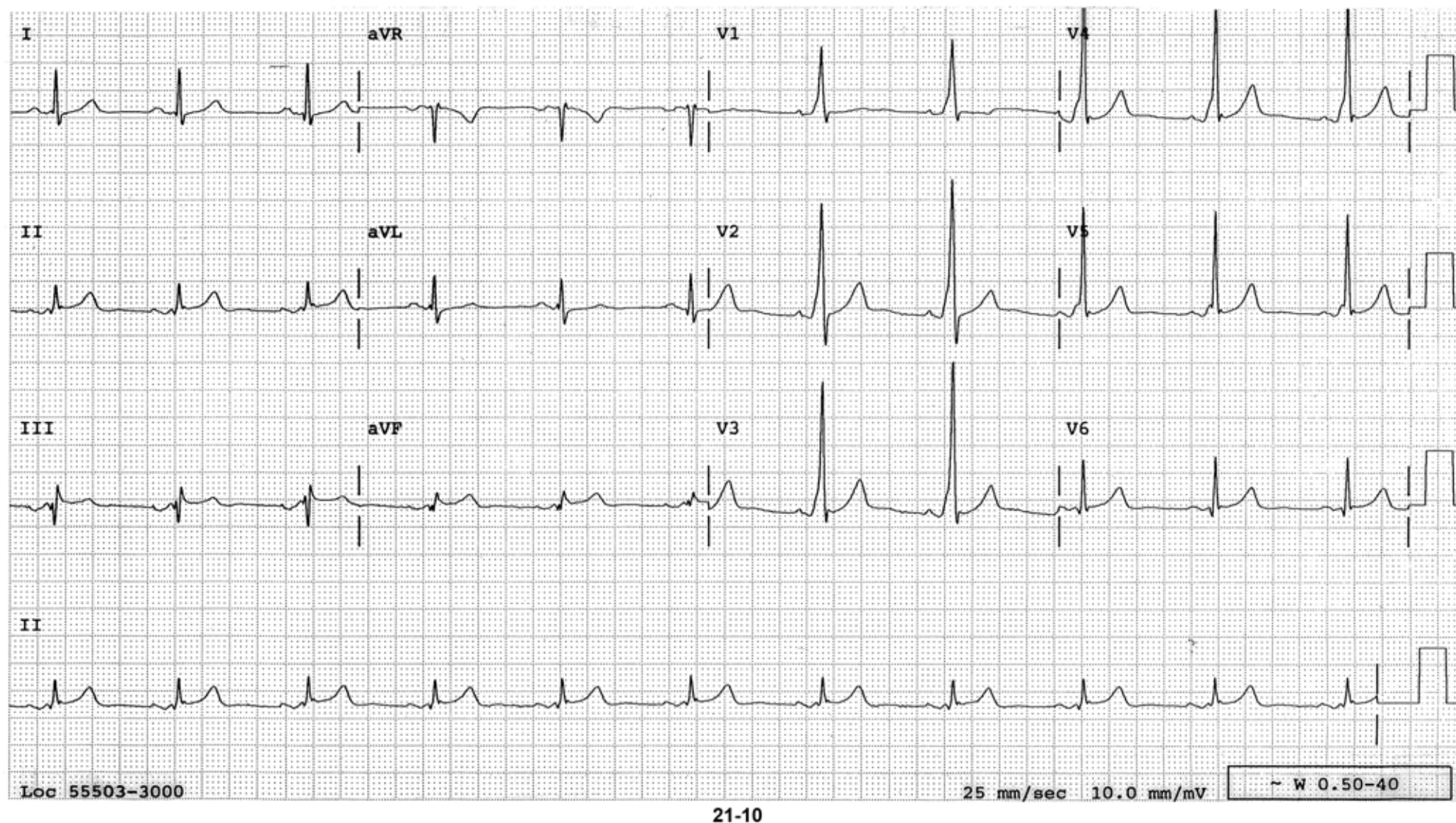
21-8 Normal sinus rhythm at a rate of 96/minute. Typical RBBB is present, manifested as broad S wave in leads I, aVL and V₆ and the second "rabbit ear" of rsR' in V₁-V₃. The first "rabbit ear" is taken away by an AMI. Judging from ST elevation in V₂-V₄, this is an acute infarction.

- Dx:
1. NSR
 2. RBBB
 3. Acute AMI



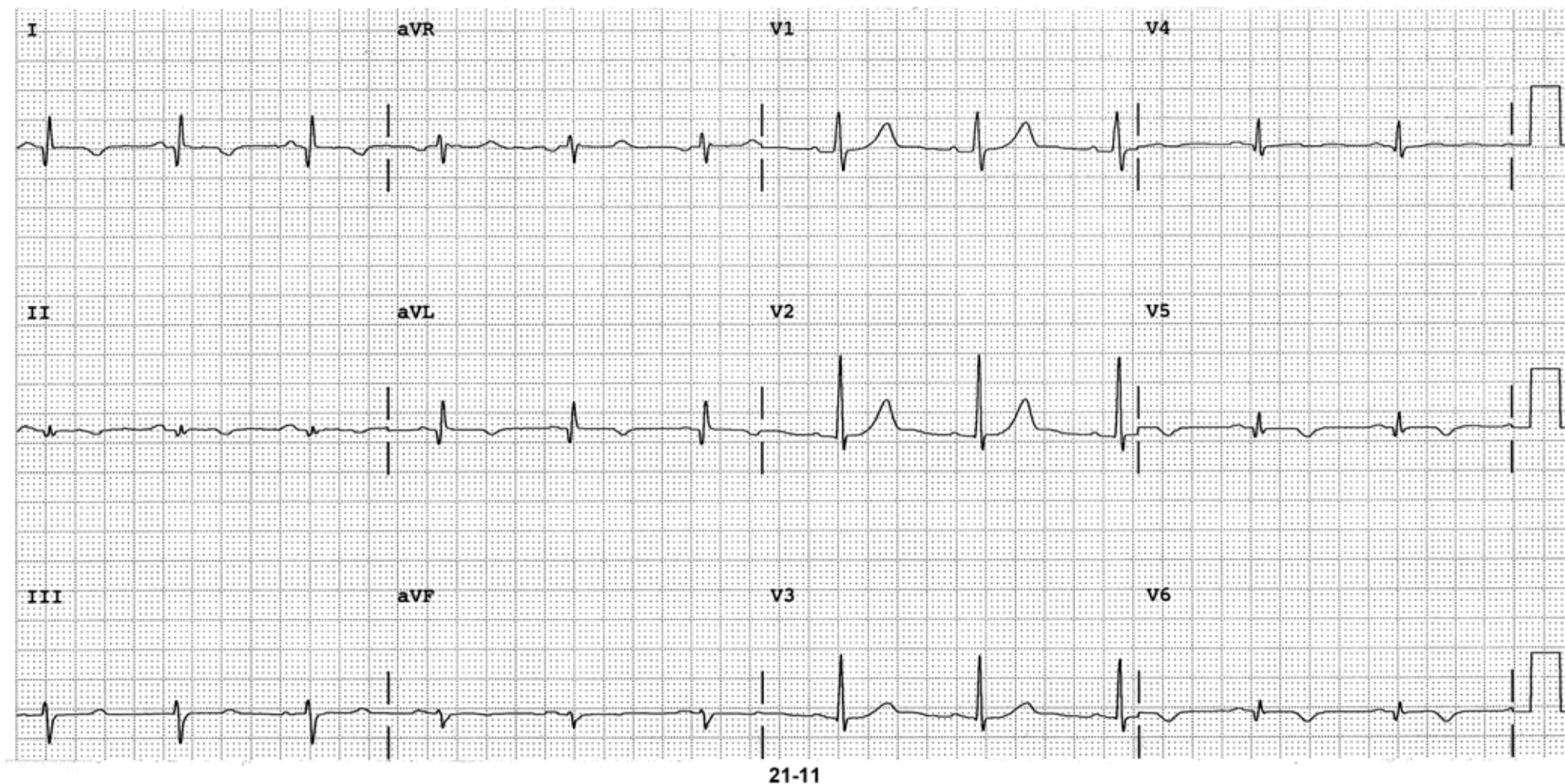
21-9 Dx: Reversed precordial leads

Clues: The P waves are biphasic in V_6 and are entirely positive in V_1 proving that these leads are reversed. Actually, judging from the R wave progression, the entire precordial leads are reversed. The upside-down P, QRS and T waves in lead I also indicate reversed arm leads as well. However, this is not an example of dextrocardia. In dextrocardia, the R waves are not tall in the right precordial leads. Findings of acute anterior infarct are present.



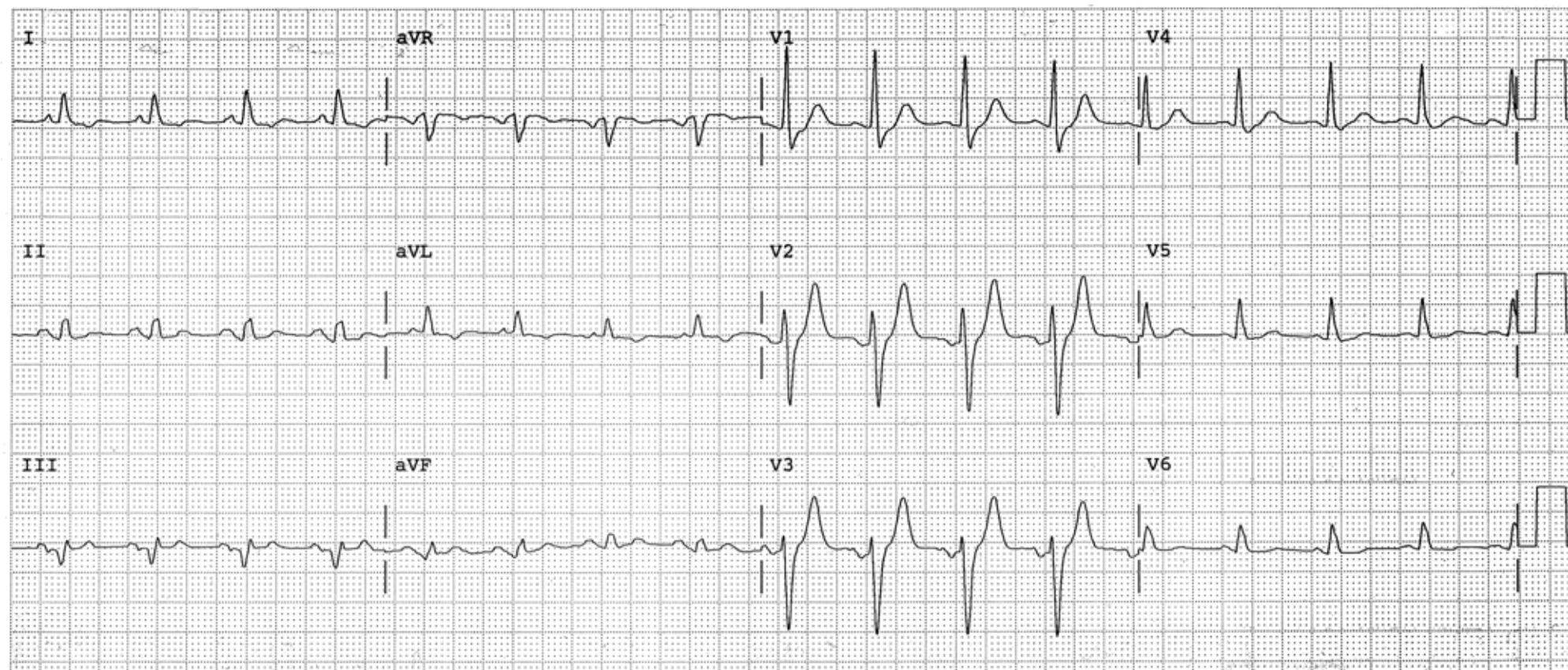
21-10 Dx: WPW syndrome

Clues: Short P-R interval and delta waves are obvious in the precordial leads. This tracing illustrates that not every lead will have a short P-R interval and a delta wave in WPW syndrome if the delta wave is isoelectric in that lead (see lead I especially). RVH should accompany RAD and S waves in the left precordial leads, which this tracing does not.



21-11 Dx: Posterolateral infarct

Clues: The lack of RAD and S wave in V_6 rules out RVH. The P-R interval is not short in any of the twelve leads, and there are no delta waves, ruling out WPW syndrome. The P wave in V_1 is slightly biphasic, and they are entirely upright in other precordial leads, indicating that the precordial leads are placed appropriately. Q waves with a T wave inversion in leads I, aVL and V_5 and V_6 indicate posterolateral MI as the cause of tall R waves in the right precordial leads.

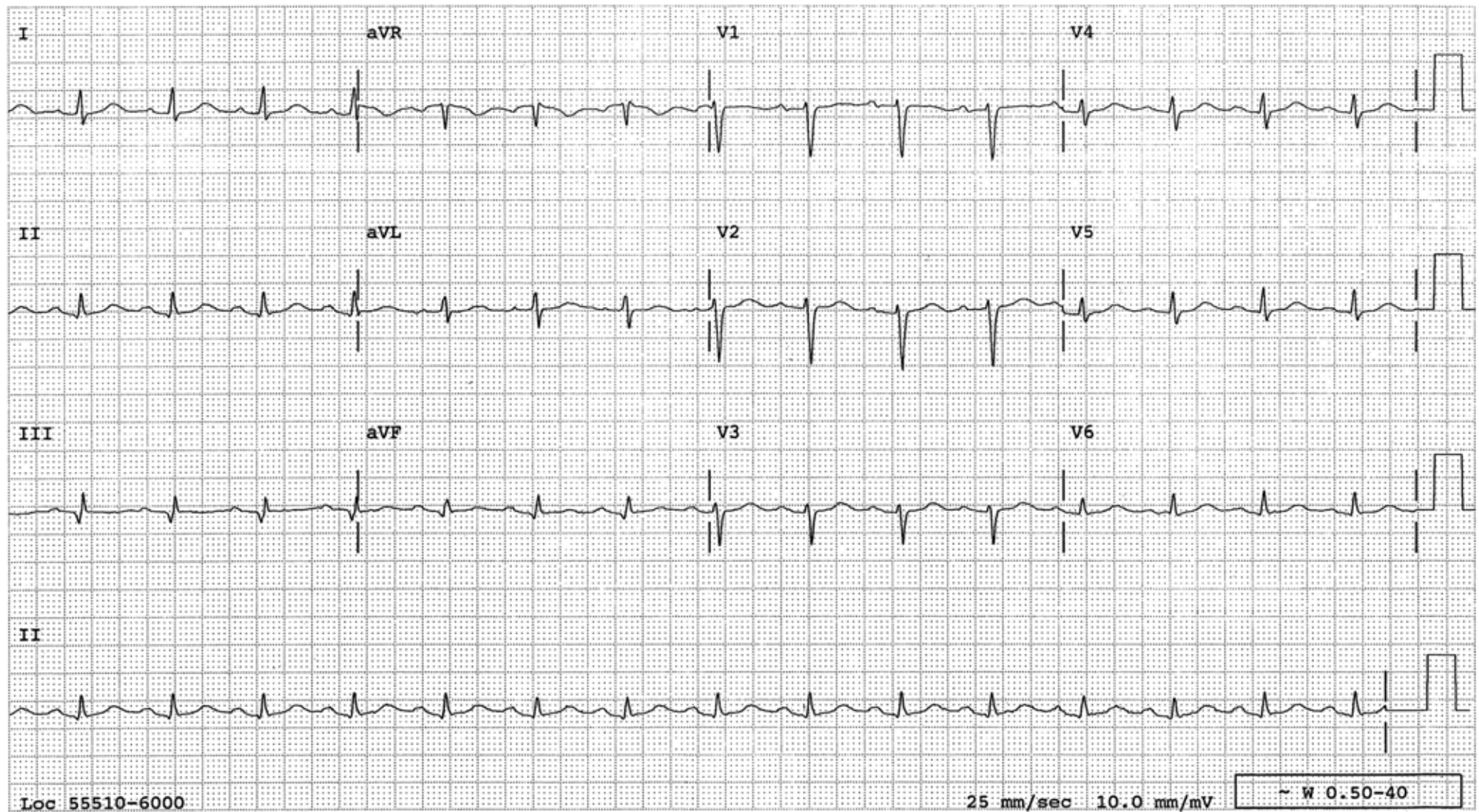


21-12

21-12 Dx: Inferior infarct and reversed V_1 and V_3 leads

Clues: An old inferior MI is evident. The R wave in V_1 is tall, raising the possibility of posterior wall involvement. However, the R waves regress rather than progress from V_1 through V_3 , suggesting that the V_1 and V_3 leads are reversed. A proof that is actually what happened is provided by the P wave morphology. The P wave in V_3 is mostly negative after a tiny positive deflection and is very good for a LAE pattern, which should be best seen in V_1 , not in V_3 . No RAD, no S wave in $V_6 \Rightarrow$ no RVH. The P-R interval is relatively short in the left precordial leads but there are no delta waves, ruling out WPW syndrome.

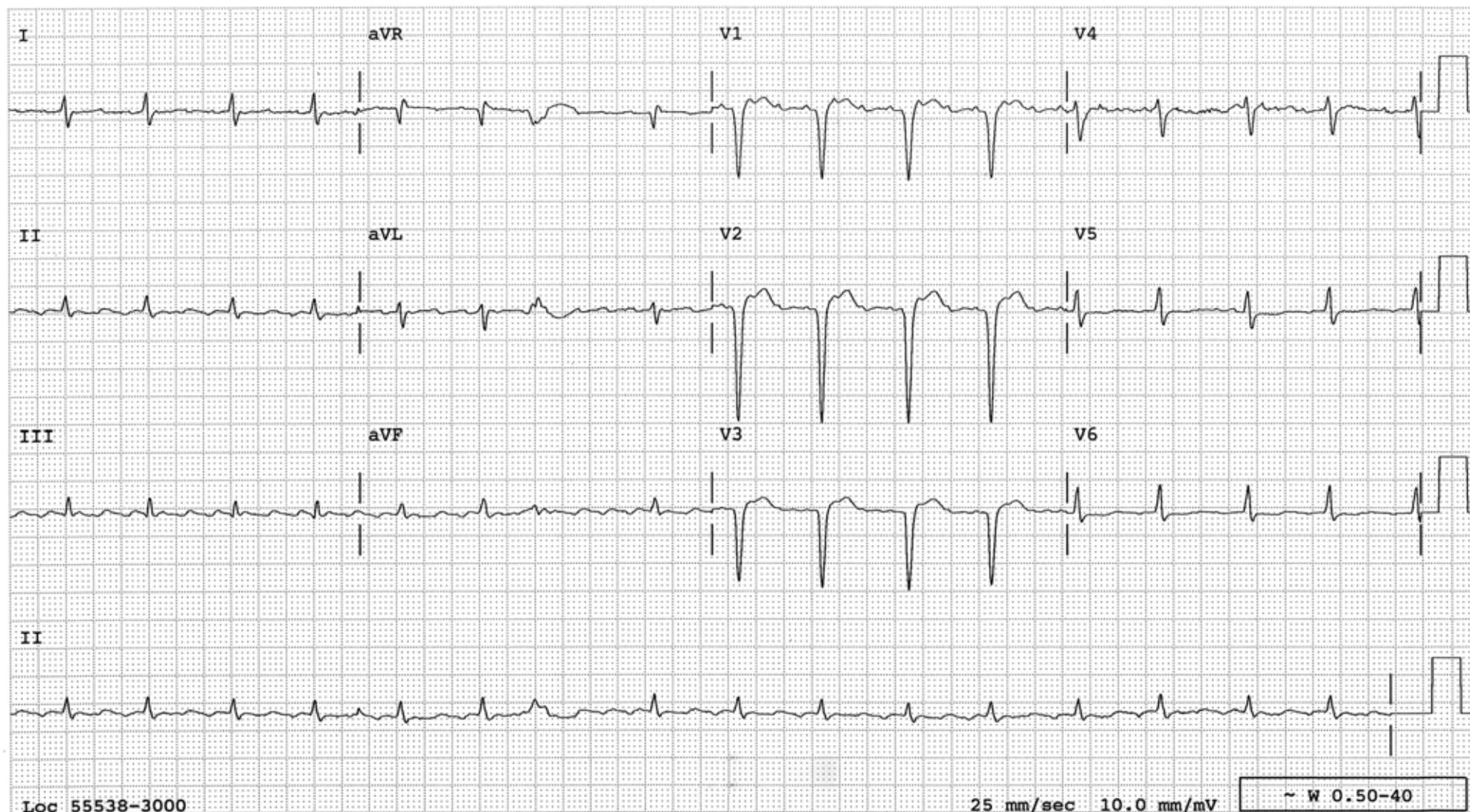
SECTION 22



22-1

22-1 Normal sinus rhythm at a rate of 91/minute. There is a Q wave in lead III which is not that impressive. Nevertheless it is wide enough and deep enough to qualify for pathologic Q wave, indicating old inferior MI.

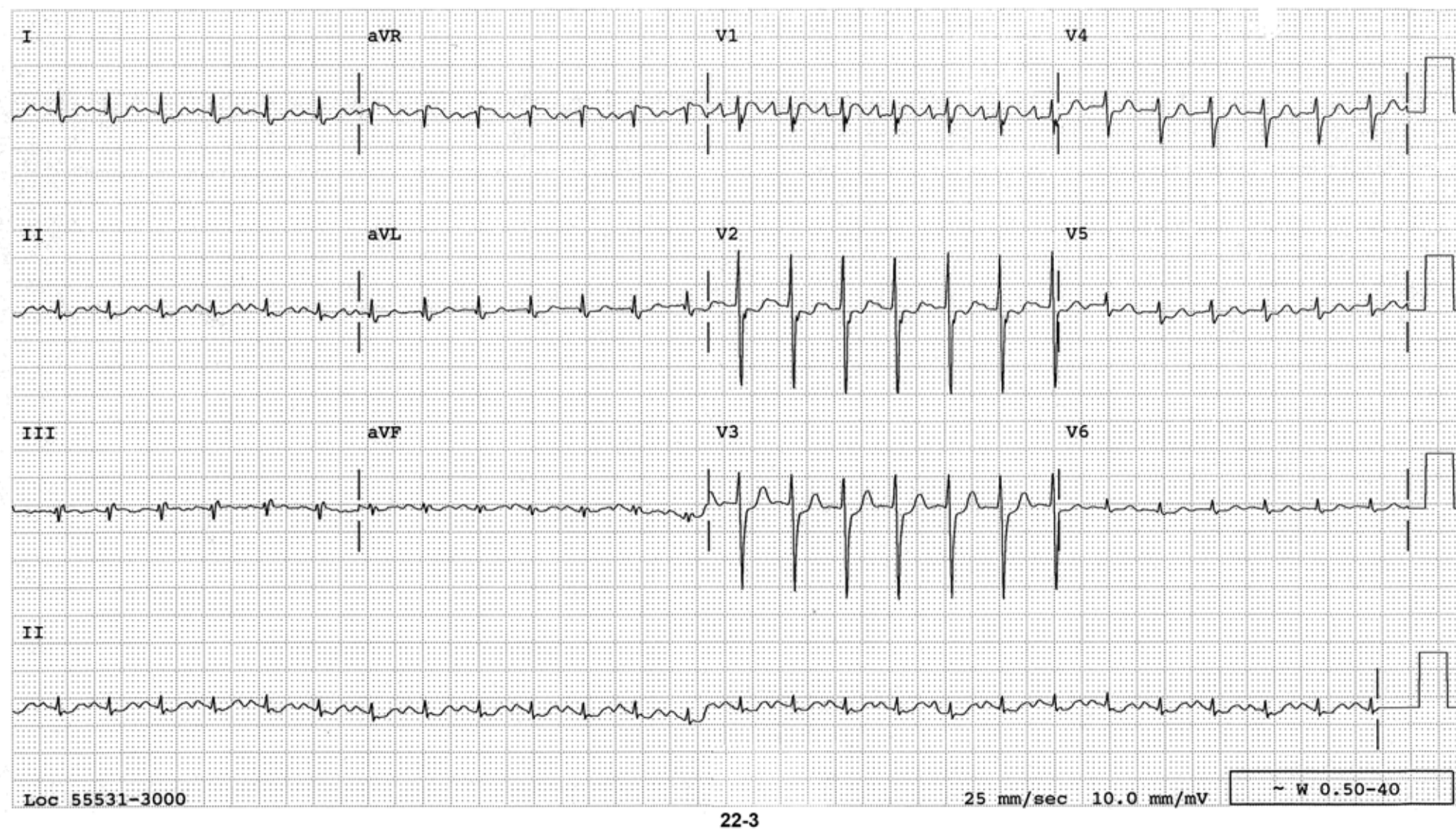
- Dx: 1. NSR
2. Old inferior MI



22-2

22-2 Regular rhythm at a rate of 100/minute. In V_1 , three atrial activities are noted between the QRSs, which occur regularly at a rate of about 300/minute. In the inferior leads, very blunt "sawtooth" pattern of atrial flutter is evident. This is more obvious in the rhythm strip during the compensatory pause after a PVC. QS pattern with ST elevation in V_1 - V_3 indicates anterior MI, probably recent. However, if this ST elevation is chronic, one has to consider the possibility of ventricular aneurysm.

- Dx:
1. Atrial flutter with 3:1 AV conduction
 2. One PVC
 3. AMI, either recent, or left ventricular aneurysm if old



22-3 Narrow QRS tachycardia at a rate of 157/minute. There is a P wave in front of each QRS. The P wave is biphasic in V_1 and is positive in lead II and is consistent with a sinus rhythm.

Dx: Sinus tachycardia



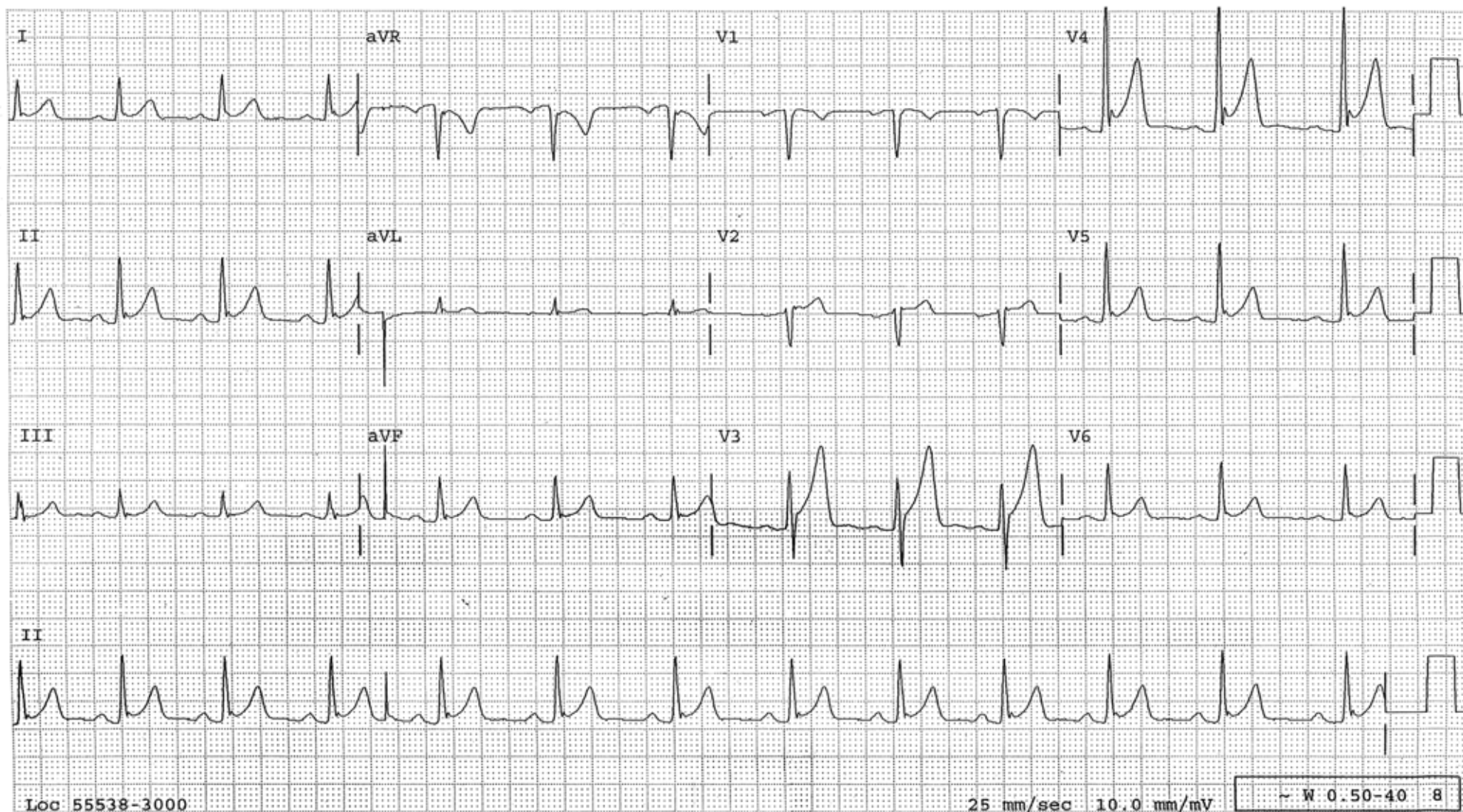
22-4 Sinus rhythm is evident in the first part of the limb leads as well as the first half of the precordial leads. In between, the rhythm reveals short runs of atrial fibrillation. Each time, the first QRS is aberrantly conducted because it is preceded by a long R-R interval (Ashman's phenomenon). The QRS voltage is abnormally low in the limb leads.

- Dx:*
1. NSR with intermittent atrial fibrillation
 2. Occasional aberrant conduction (Ashman's phenomenon)
 3. Low QRS voltage in the frontal leads



Dx: 1. NSR

2. Normal ST elevation

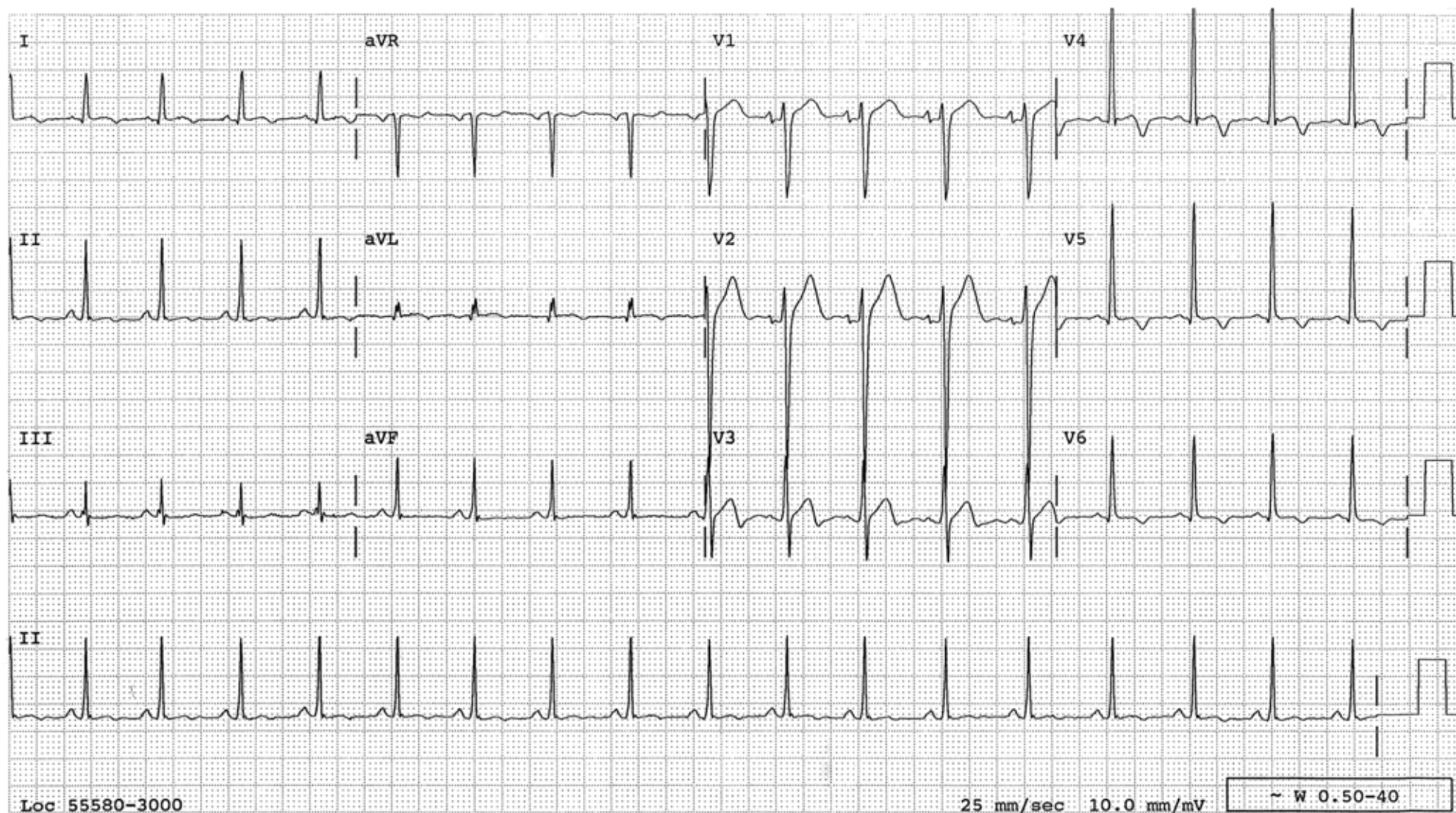


22-6

22-6 Sinus rhythm at a rate of 80/minute, ST-segment is elevated in mid precordial leads. The ST-segment is concave. There is a notch at the J point (junction between the QRS and ST-segment) in V_4 . T waves are upright and tall. These are all the features of early repolarization pattern as a normal variant.

Dx: 1. NSR

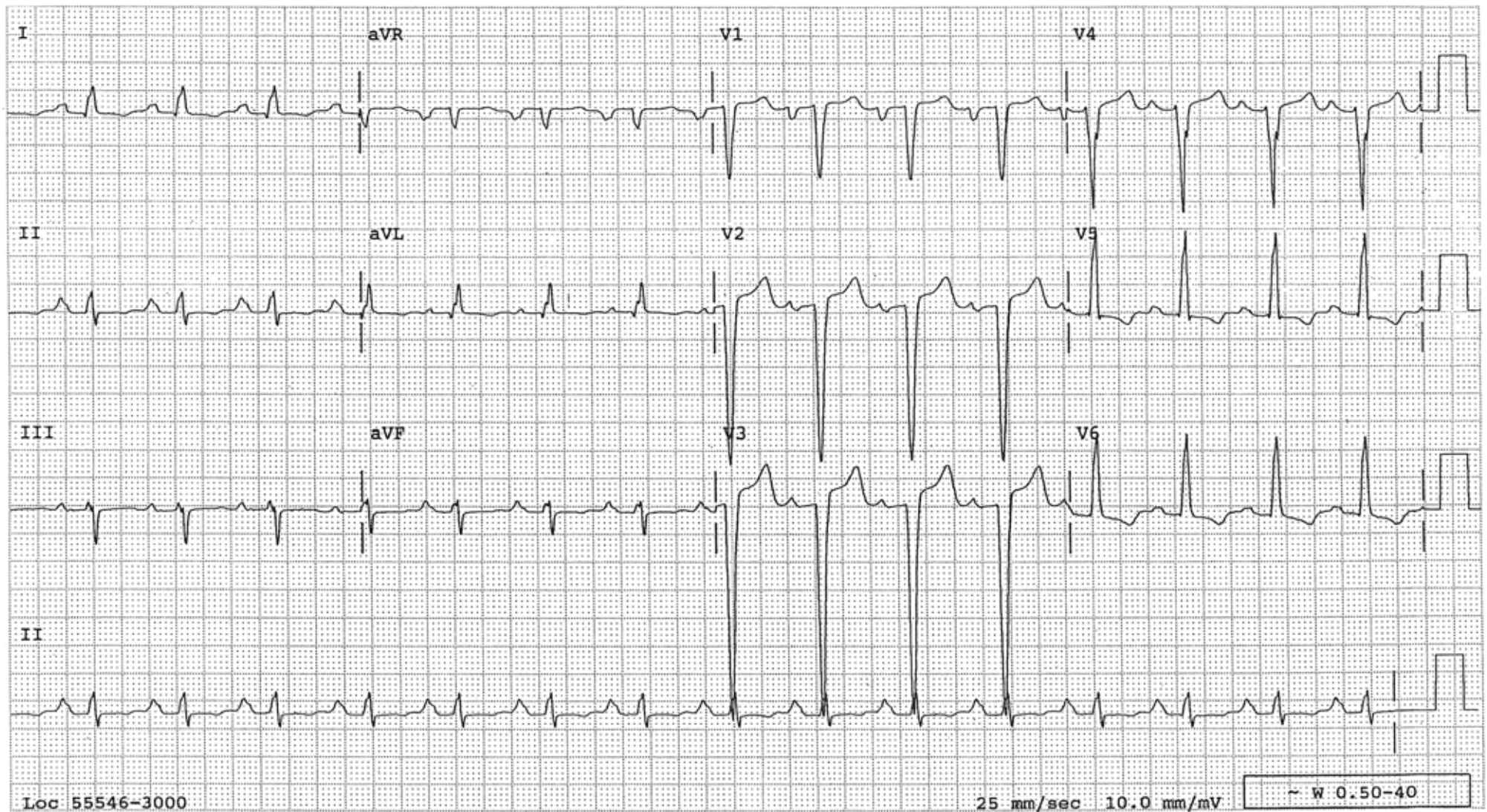
2. Early repolarization pattern as a normal variant



22-7

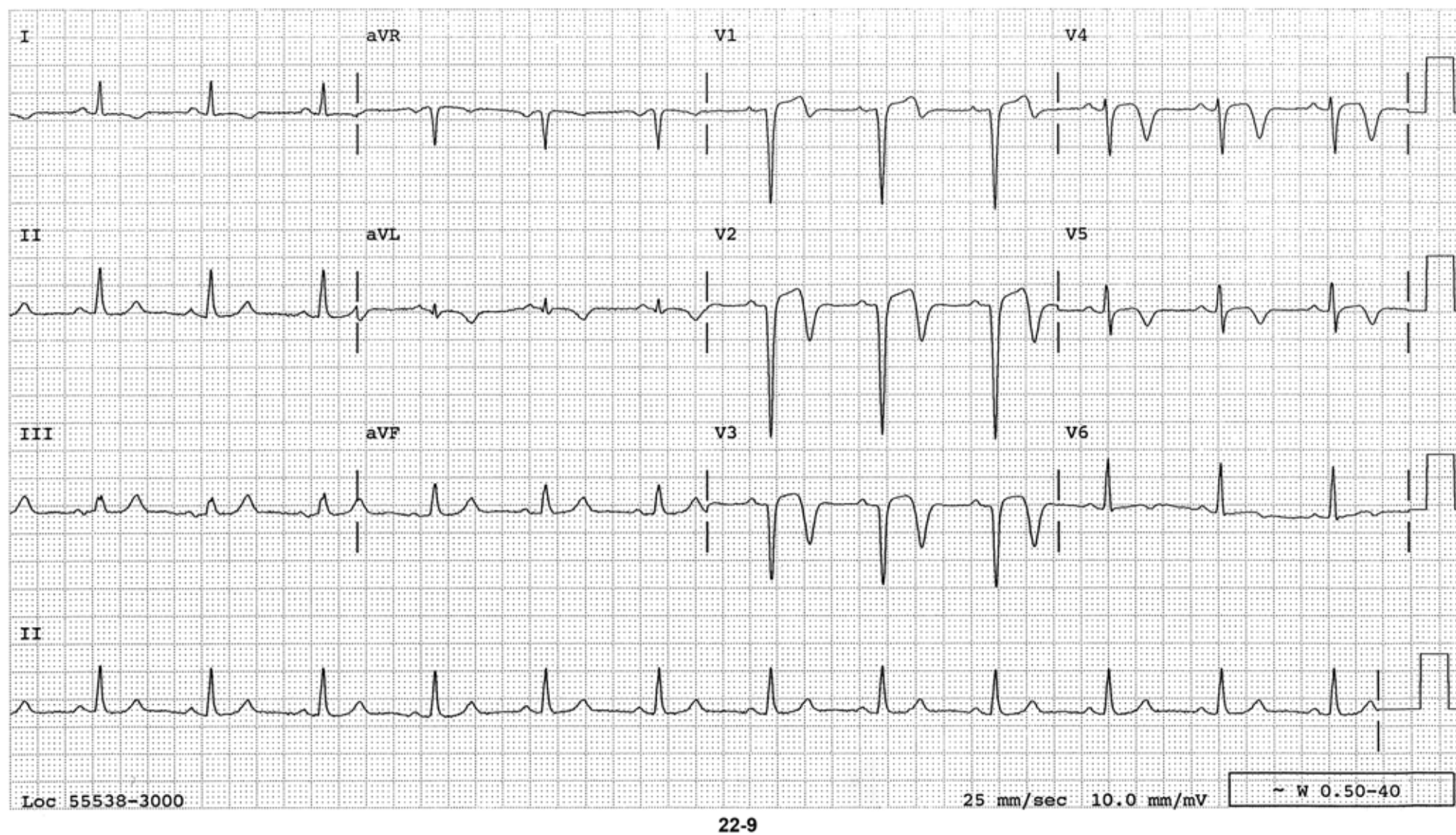
22-7 This is another form of ST-elevation as a normal variant. ST-segments are elevated in mid-precordial leads. There is a notch, albeit small, at the junction in V_4 , which is necessary for this Dx just as in early repolarization pattern. This entity is distinctly different from the early repolarization pattern in that the T waves are upright and tall in the latter while they are inverted in this entity. The ST-segment tends to be coved, while it is concave in the early repolarization pattern. The Q-T interval tends to be short. Limb leads can also be involved. These ECG findings certainly can be mistaken for acute pericarditis or infarction if one is not aware of this entity. This entity occurs almost exclusively in young black men.

Dx: ST Elevation of "The Other" normal variant



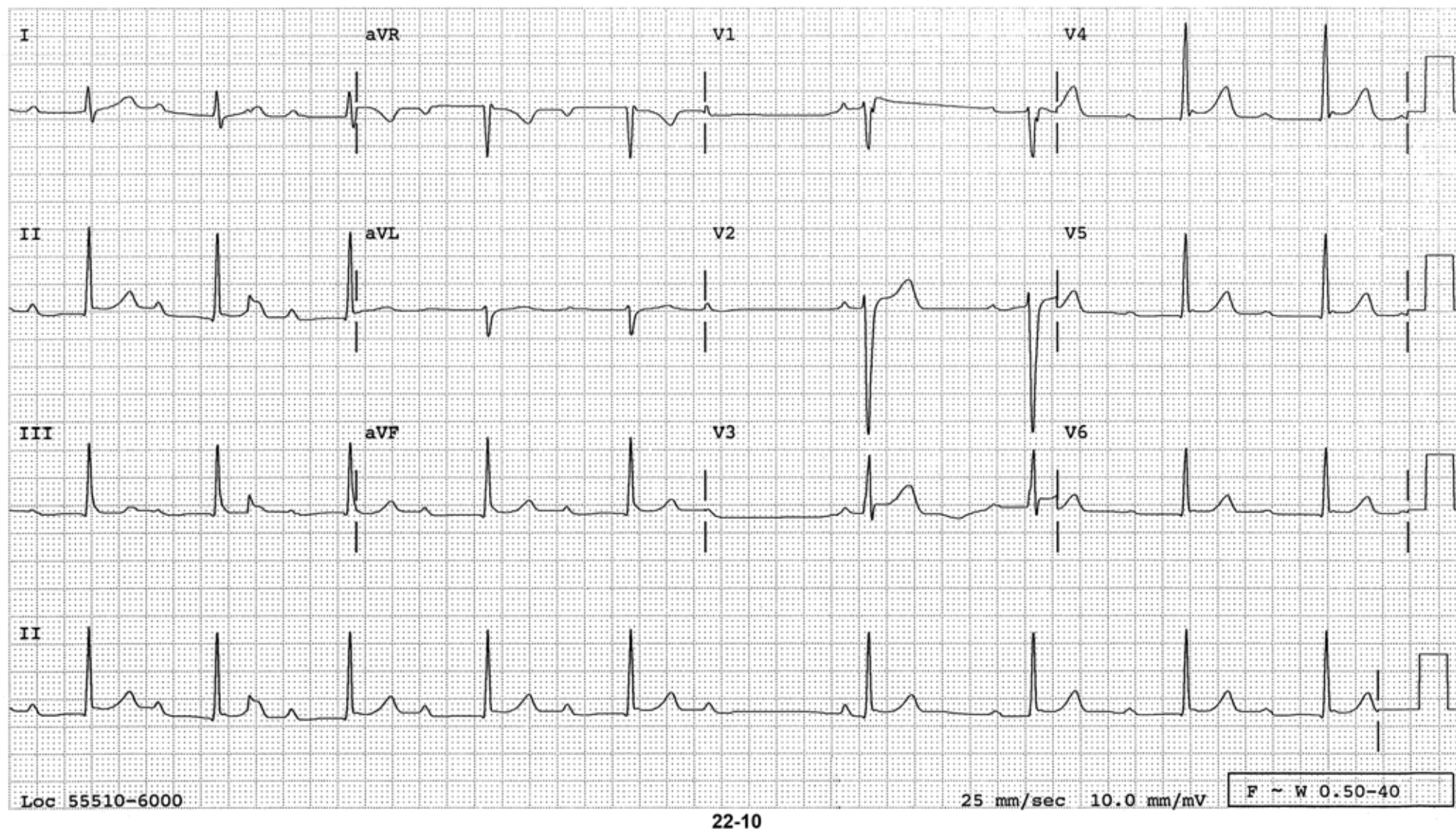
22-8 Normal sinus rhythm at a rate of 90/minute. Increased QRS voltage in the precordial leads, ST-T changes in V_5 - V_6 and LAE are all diagnostic of LVH. The QS pattern with concave ST-segment elevation in V_1 - V_3 is all from LVH rather than from an acute anteroseptal MI. Acute MI would more likely have caused convex ST-segment ending in an inverted T wave. In LVH, on the horizontal plane, not only the QRS vector loop becomes bigger but it swings posteriorly with no electrical force coming anteriorly. This results in QS pattern in V_1 - V_3 . LVH manifesting like this is one of the most common tracing mistaken for an acute MI.

- Dx: 1. Sinus rhythm
2. LVH



22-9 Normal sinus rhythm at a rate of 74/minute. QS pattern in the right precordial leads with a slight elevation of the ST-segment and a terminal T wave inversion reflect recent AMI.

- Dx:*
1. NSR
 2. Recent anteroseptal infarct

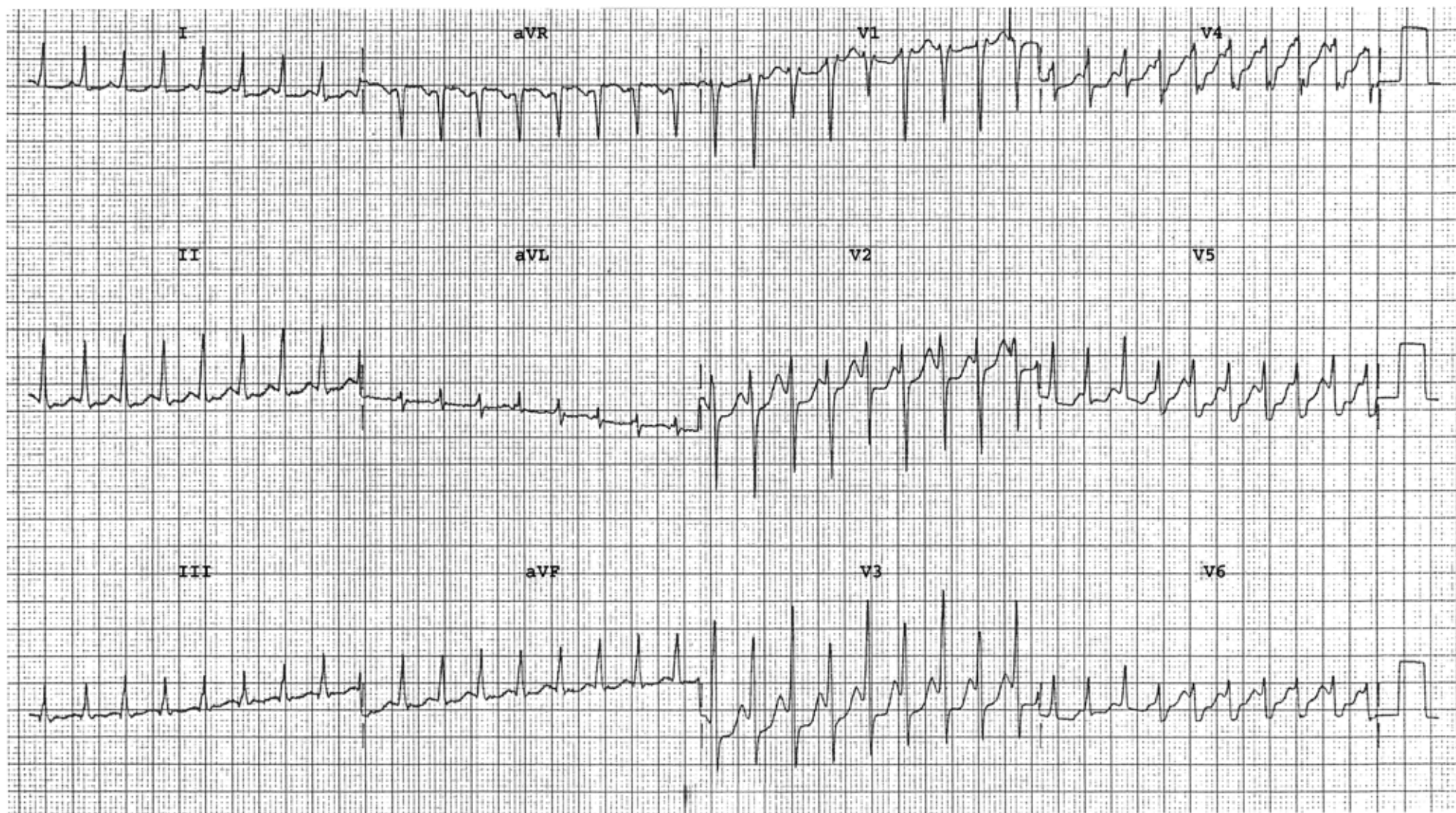


22-10 P waves occur regularly at a rate of 58/minute. In the middle of the strip, one P wave is blocked. Following this, the P-R interval gradually lengthens, revealing a typical AV Wenckebach phenomenon. As demonstrated in this tracing, in AV Wenckebach phenomenon, the progressively lengthening PR interval is most noticeable at the beginning, not near the end of the cycle; i.e. PR interval progressively lengthens with decreasing increments. That is the reason why the first R-R interval is a bit longer than others. Another feature of Wenckebach phenomenon: the pause from the blocked P wave is always shorter than two conducted R-R intervals because the pause is encroached upon by the delayed QRS. These secondary features of Wenckebach phenomenon (the first RR interval being a bit longer than others and the pause being shorter than two conducted RR intervals) are not important in dealing with A-V Wenckebach phenomenon because the diagnosis is readily made just from the progressive lengthening PR interval. However, they come in handy in appreciating Wenckebach phenomenon at other conduction levels of rhythms such as SA block or atrial flutter or AV junctional rhythm with Wenckebach phenomenon.

Dx: Sinus rhythm with AV Wenckebach phenomenon (Mobitz Type I 2° AV block)

22-11 *Question:* This tracing is a proven case of AV junctional reentrant tachycardia, which is the most common form of SVT.

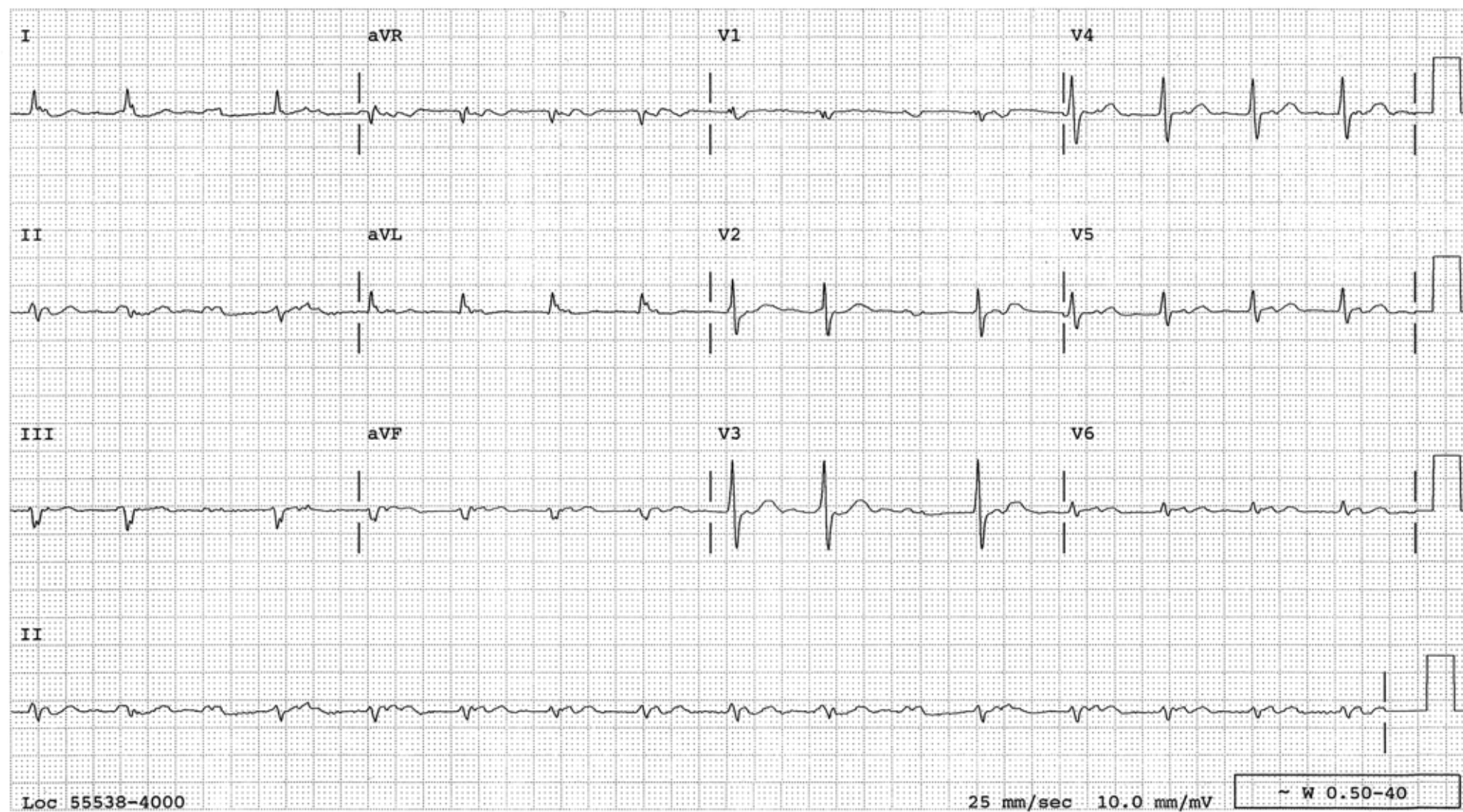
1. The electrical alternans seen in several leads (aVL, V₁, V₂, and V₃) should make one think of cardiac tamponade.
☐ Yes ☐ No
2. The electrical alternans in this case is most likely accompanied by pulsus alternans.
☐ Yes ☐ No
3. The horizontal ST depression seen V₃ is highly indicative of myocardial ischemia.
☐ Yes ☐ Not necessarily
4. The QRS are widened out in the latter part of the tracing reflecting (choose one from below):
☐ (a) Aberrant conduction
☐ (b) Transformation of SVT to VT
☐ (c) It is the ST depression which makes the QRS look wider



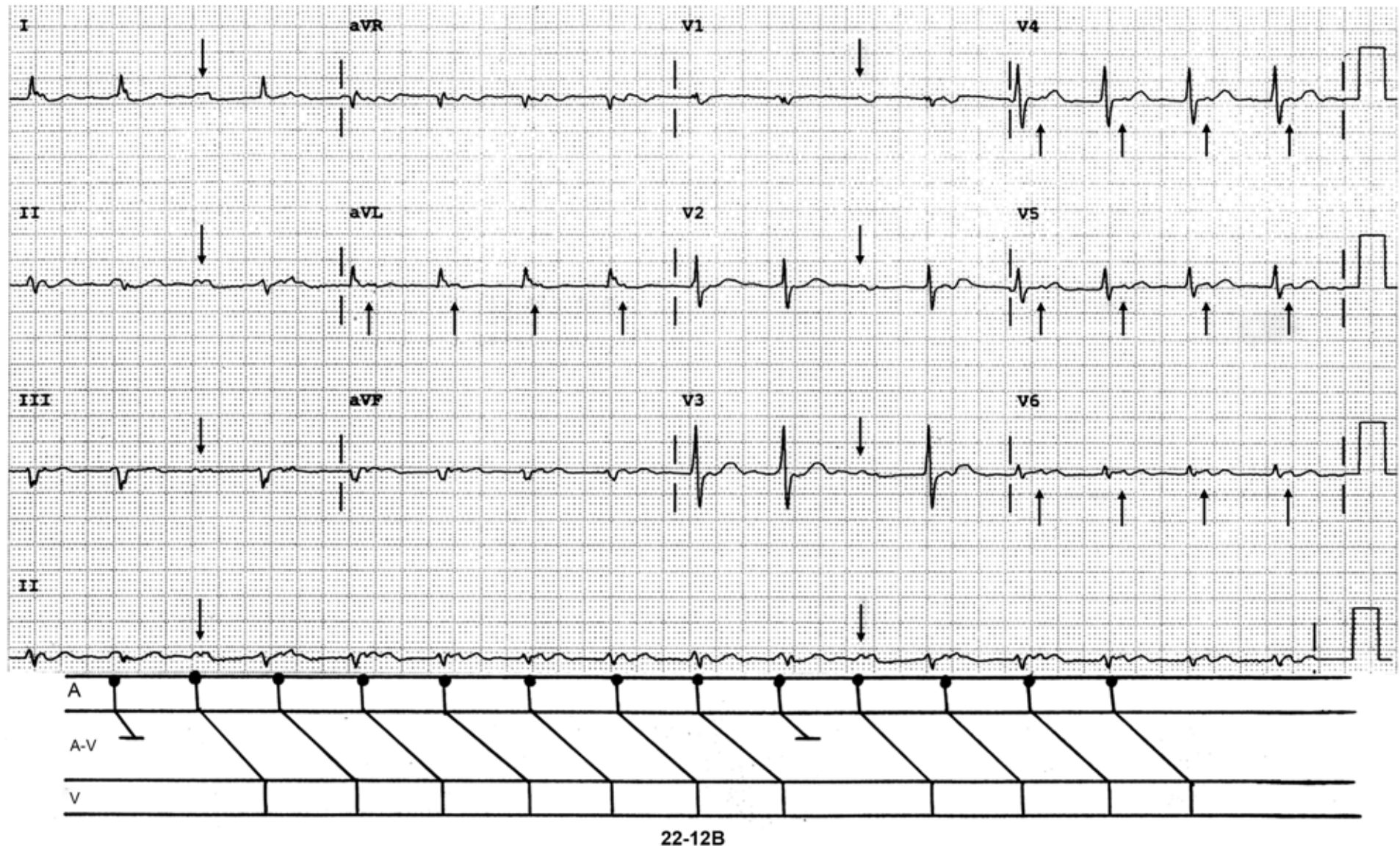
22-11

- Answer:*
1. No
 2. Yes
 3. Not necessarily
 4. (a) Aberrant conduction

Discussion: Electrical alternans is well-known to occur in some cases of SVT, atrial flutter or VT without cardiac tamponade. Only during sinus rhythm it implies cardiac tamponade. Additionally, the EA during SVT often is accompanied by pulsus alternans, while that during pericardial effusion or cardiac tamponade is not. SVT may cause myocardial ischemia, resulting in ST depression, but it is well-known that ST depression can occur during SVT without myocardial ischemia. The QRSs are widened in the latter part of the tracing. The fact that the rate stays the same as in the earlier part of the tracing, as well as the presence of a broad S wave, indicates RBBB-type aberrant conduction rather than a new rhythm problem.



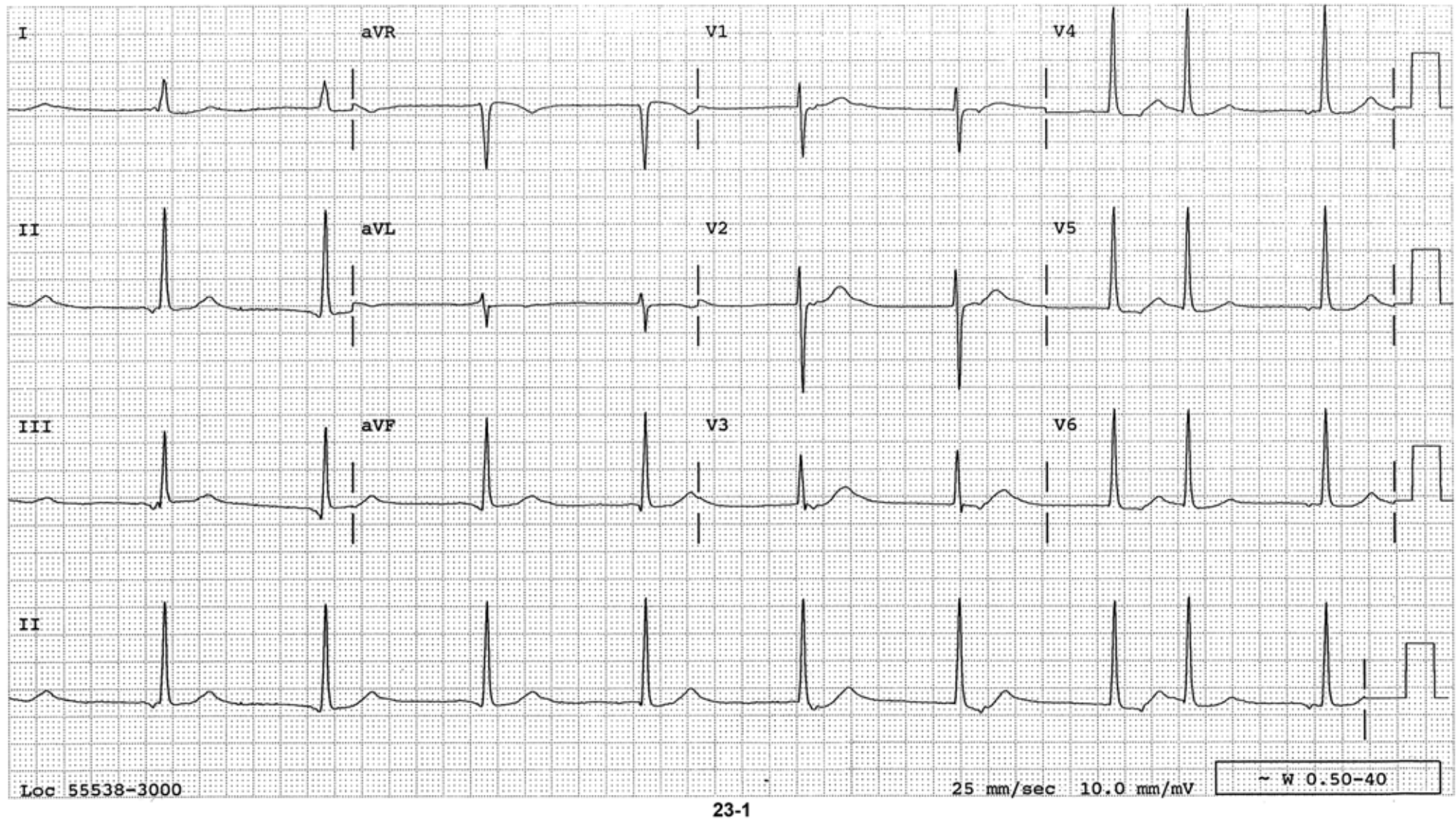
22-12A



- 22-12 P waves are not readily visible. The two blips (\downarrow) during the pauses are definitely P waves. They are broad and double humped instead of narrow and distinct in lead II, making them difficult to see when they occur on the ST segment in the rhythm strip of lead II. Left atrial enlargement is causing this judging from the fact that it is mostly negative and more than 1×1 mm in V_1 . P waves are barely but definitely visible in leads aVL and V_{4-6} (\uparrow) and they occur regularly, indicating the basic rhythm is sinus. One can appreciate the R-P interval becoming gradually shorter, which means the P-R interval is getting progressively longer. The blocked P wave times within the QRS. The pause is shorter than two R-R intervals. These observations resulted in the above ladder diagram.

Dx: AV Wenckebach phenomenon as diagrammed in Figure 22-12B in the above figure

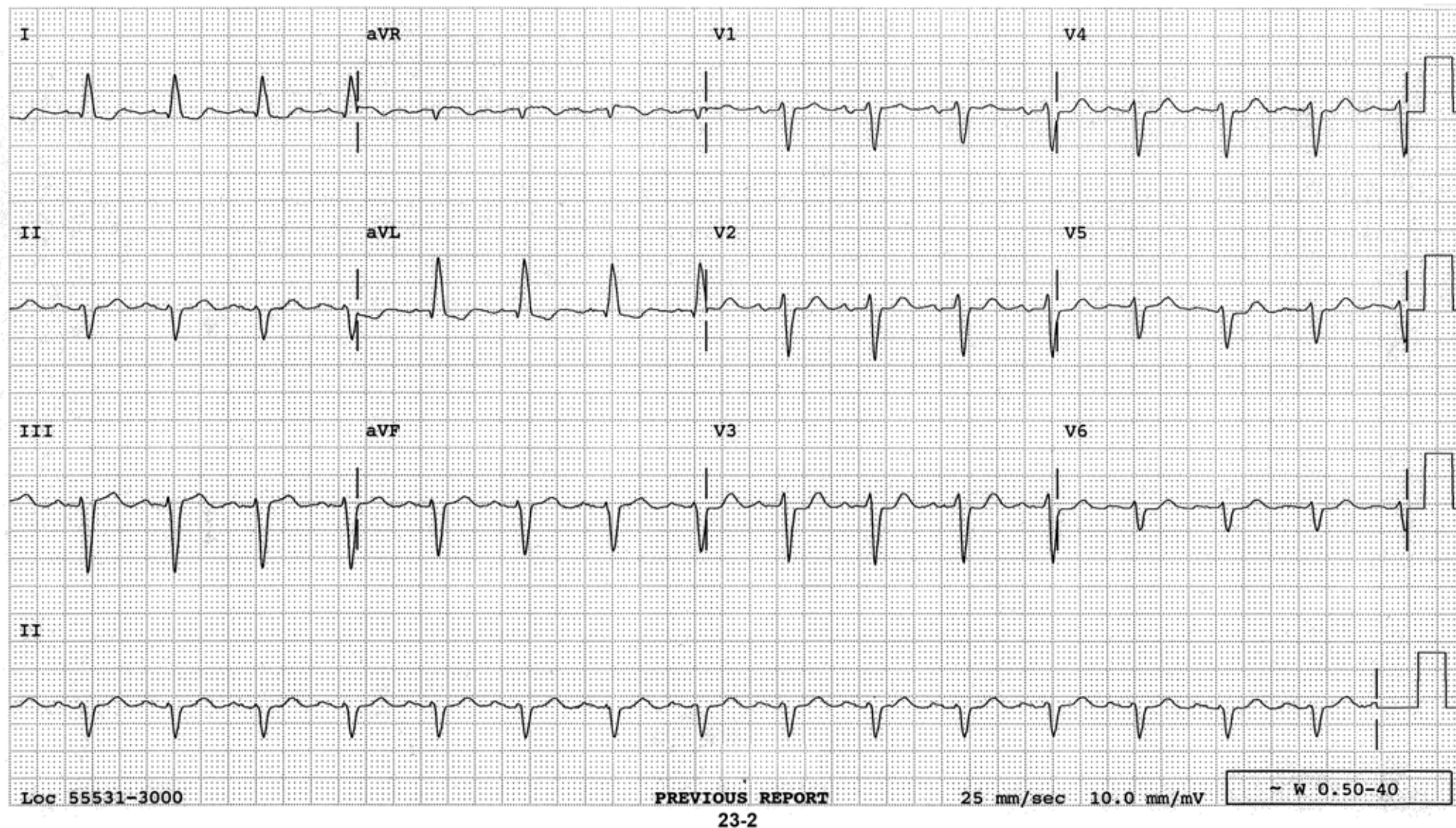
SECTION 23



23-1 No sinus P waves are seen and a junctional rhythm escapes at a rate of 53/minute. Retrogradely conducted P waves are seen either in front (the first and the last complex) or after the QRS (in the latter part of the tracing). The retrograde P wave is occurring gradually later and later and the third complex from the end has a retrograde P wave occurring considerably after the QRS, which allows the retrograde impulse to "bounce off the atrial wall", and conducts to the ventricle; hence called an "echo" (reciprocal) complex.

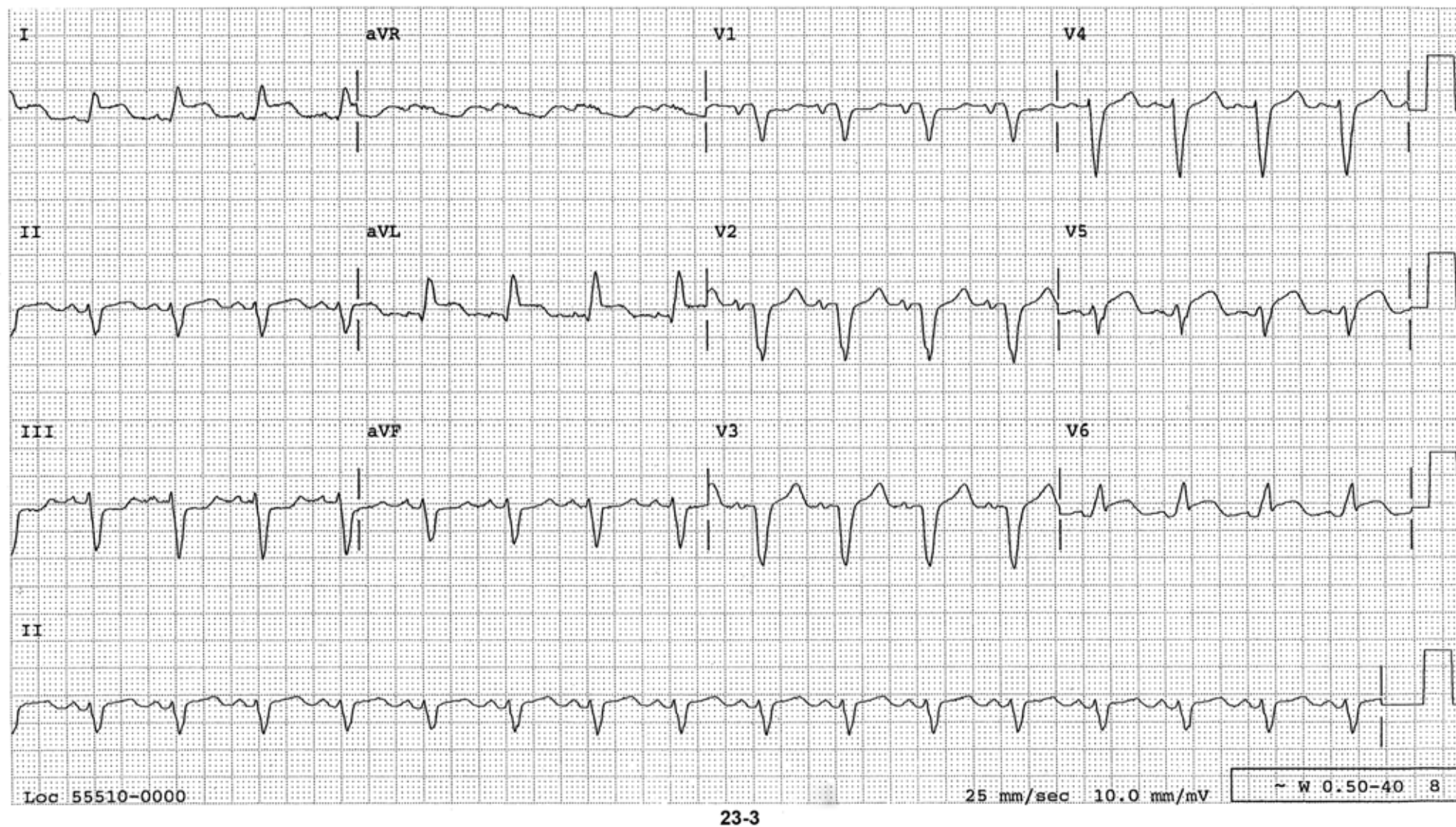
Dx: 1. Sinus node dysfunction as the primary disorder

2. Junctional escape rhythm with retrograde conduction to the atria, which gradually falls behind (retrograde J-A Wenckebach phenomenon) terminating in an echo beat as the obligatory secondary manifestations



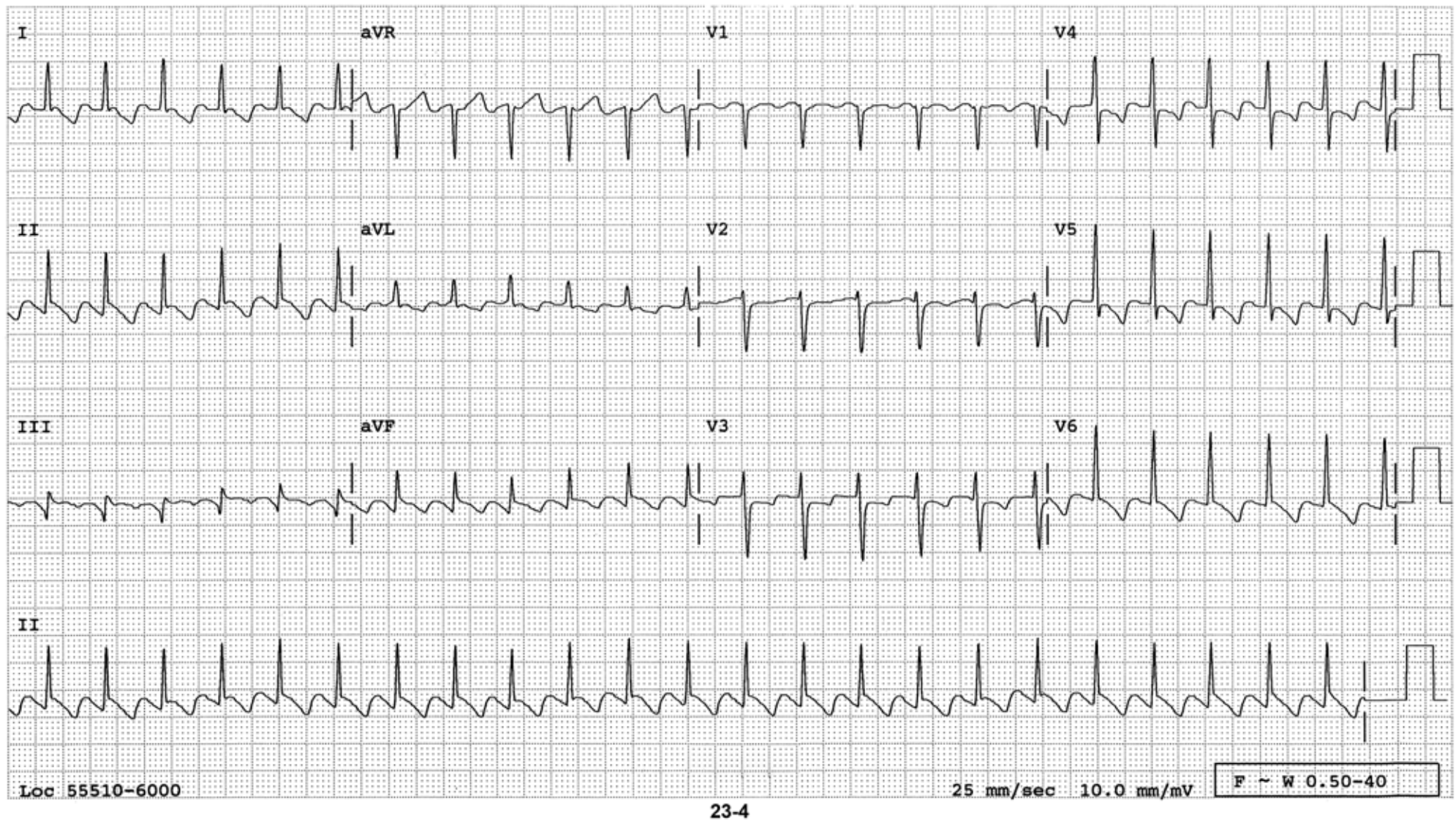
23-2 Normal sinus rhythm at a rate of 94/minute. The P-R interval is at the upper limit of normal. The QRS axis is markedly shifted to the left and is diagnostic of LAFB. In the precordial leads, the S wave is still deeper than the height of the R wave in V_6 , indicating late transition. This late transition often accompanies LAFB. The QRS is wide, measuring 116 milliseconds, reflecting IVCD.

- Dx:
1. NSR
 2. LAFB with IVCD
 3. Late transition



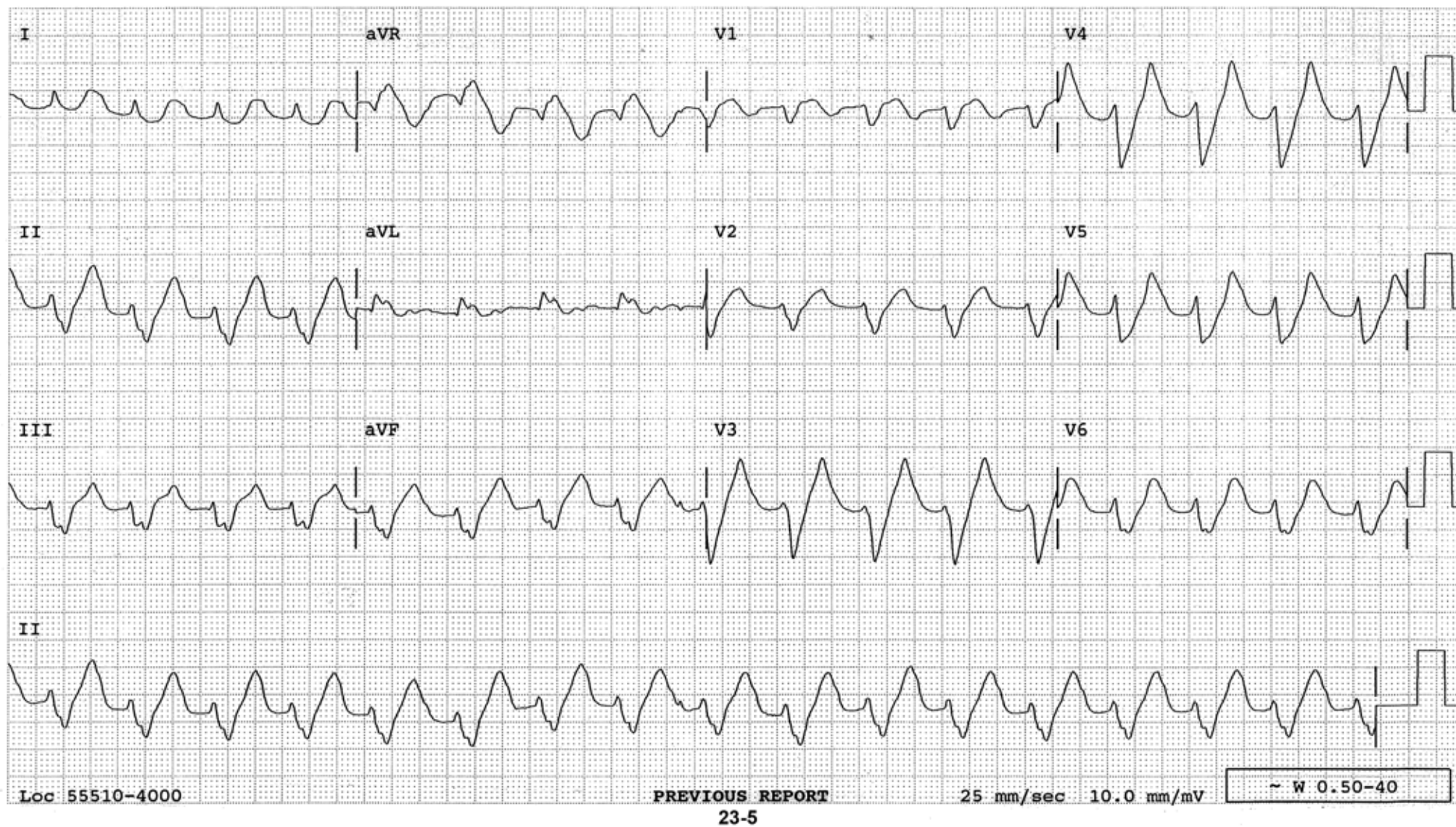
23-3 Sinus rhythm at 99/minute. QRSs are wide. Monophasic R waves in leads I, aVL and V₆, and QS pattern in V₁-V₃ are typical of LBBB. The ST-segment is deviated to the same direction as the major component of the QRS (concordant ST changes) in leads I, aVL and V₆, and is diagnostic of lateral wall STEMI. Thus, even during LBBB, STEMI can be diagnosed if there is a concordant ST deviation.

- Dx:
1. NSR
 2. LBBB
 3. Lateral STEMI



23-4 Narrow QRS regular tachycardia at a rate of 141/minute. "Sawtooth" pattern of atrial flutter is evident in the rhythm strip of lead II. Q waves in the inferior leads indicate inferior MI.

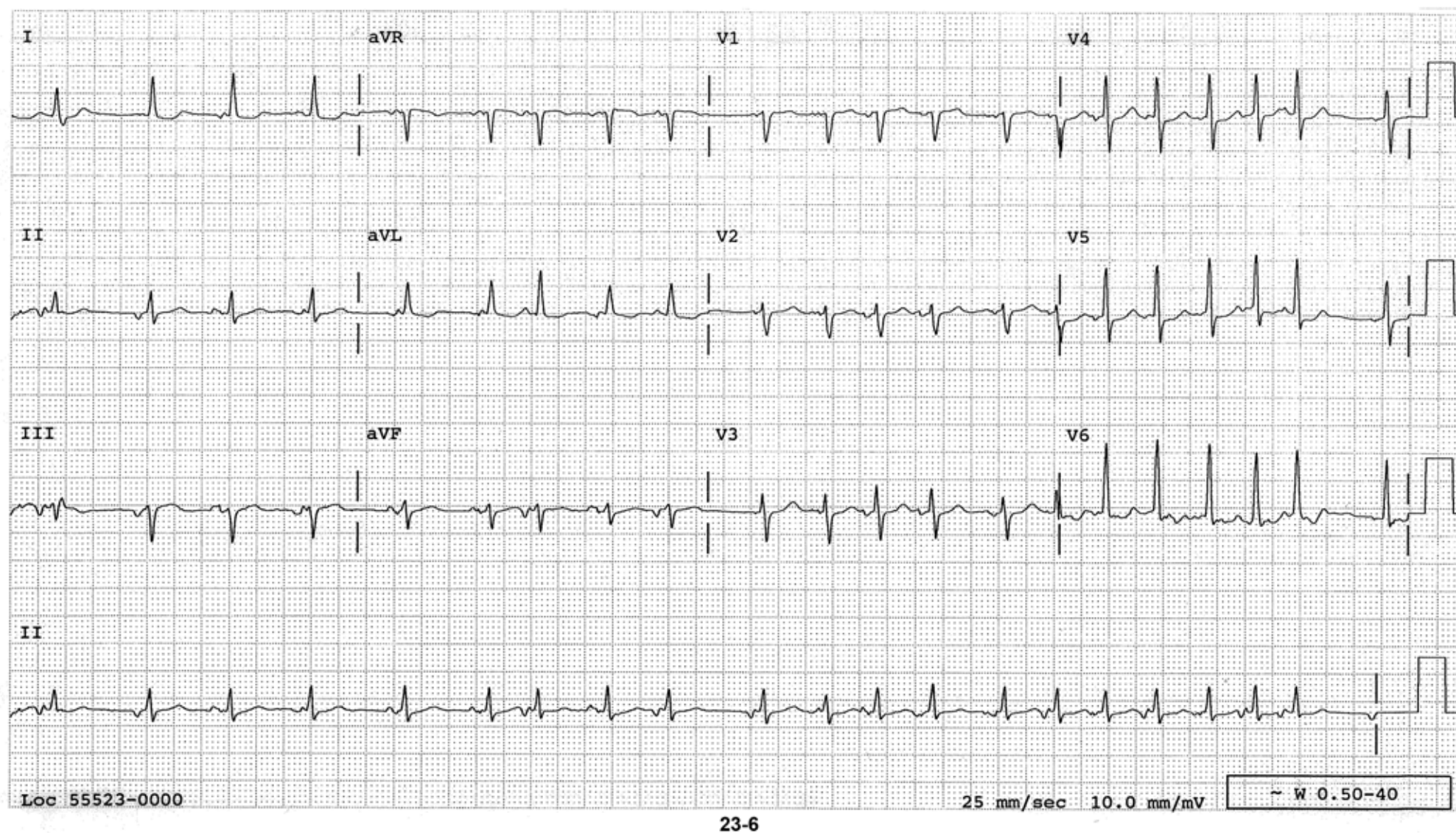
- Dx:*
1. Atrial flutter with 2:1 AV conduction
 2. Inferior infarct
 3. Diffuse ST-T changes



23-5 Wide QRS rhythm at a rate of 100/minute. No P waves are distinctly identified. The T waves are somewhat pointed and tall in V_3 and V_4 . These findings are highly suggestive of hyperkalemia (the serum potassium was 7.8 mEq/L at this time).

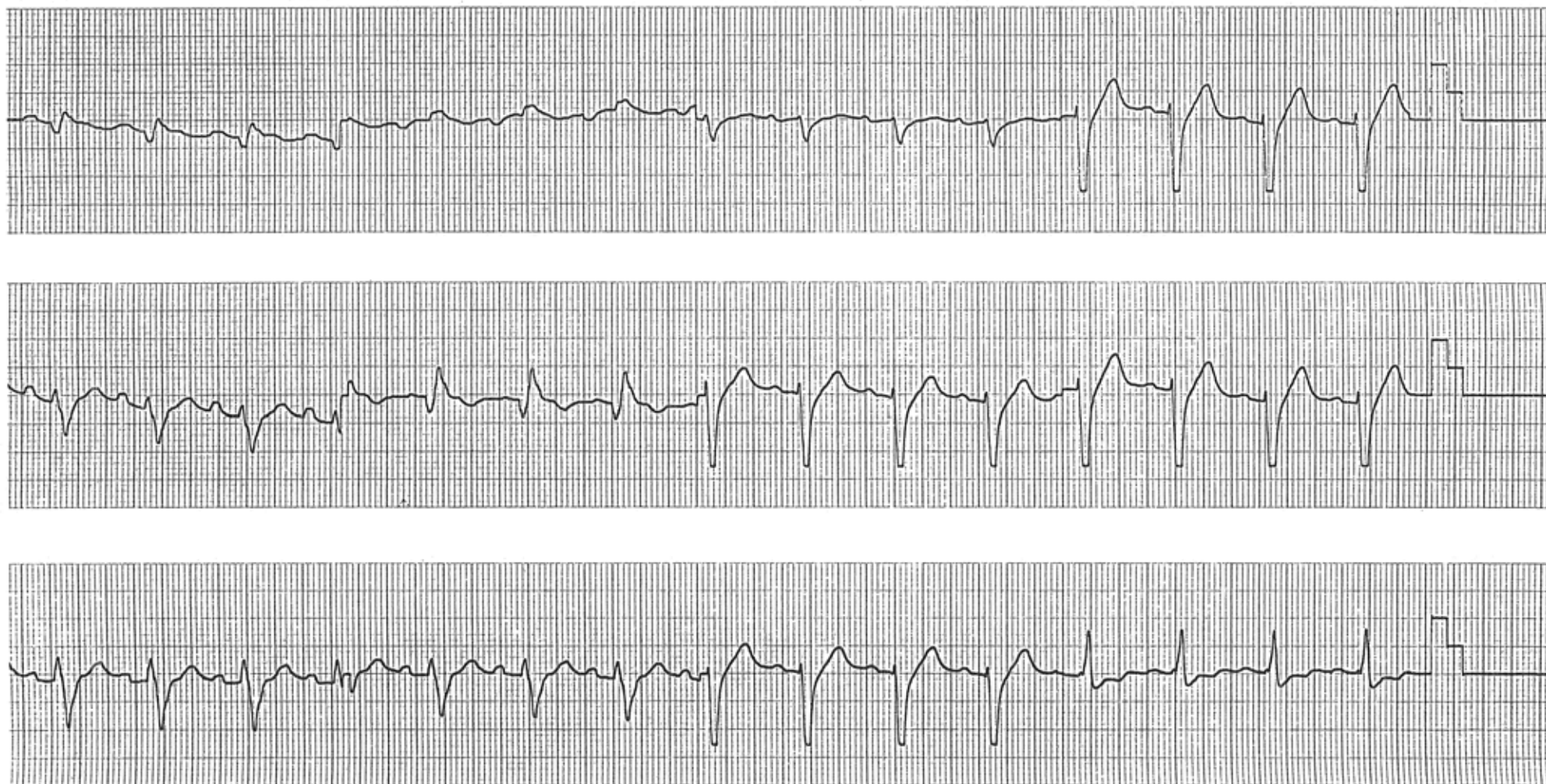
Dx: Hyperkalemia

23-6 Question: What is the rhythm especially in the latter half of the tracing?



Answer: MAT

Discussion: Irregular rhythm with an average ventricular rate of about 140/minute. Each QRS is preceded by a P wave which occurs irregularly with a changing morphology. These findings are diagnostic of MAT.



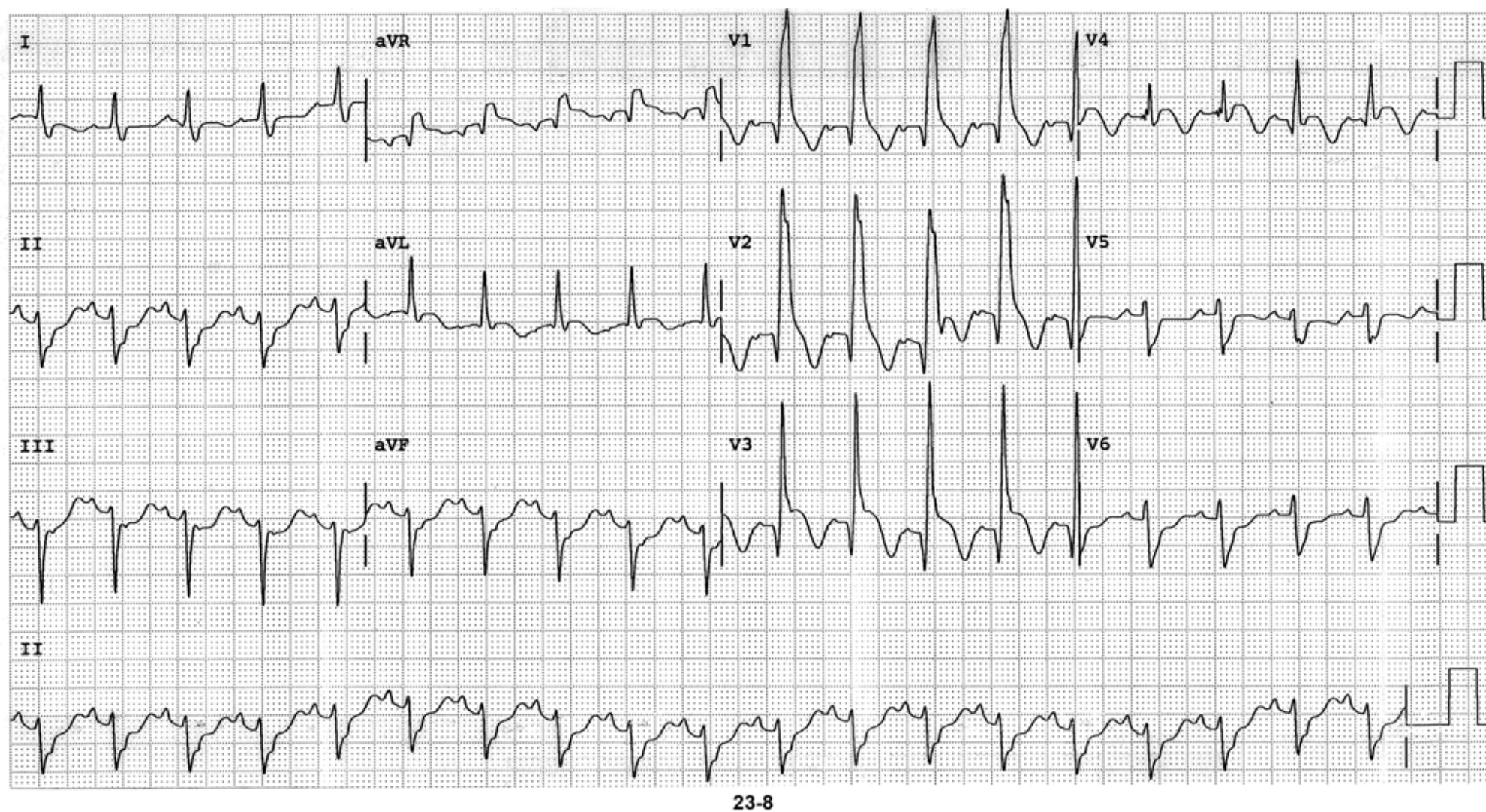
23-7

23-7 Sinus rhythm at a rate of 95/minute. The QRS is wide and measures about 160 milliseconds, reflecting IVCD of LBBB type. Q waves are very wide in leads 1 and aVL, diagnostic of high lateral infarction.

- Dx:*
1. Sinus rhythm
 2. IVCD (atypical LBBB)
 3. High lateral infarct

23-8 Question: This tracing reveals (choose one from below):

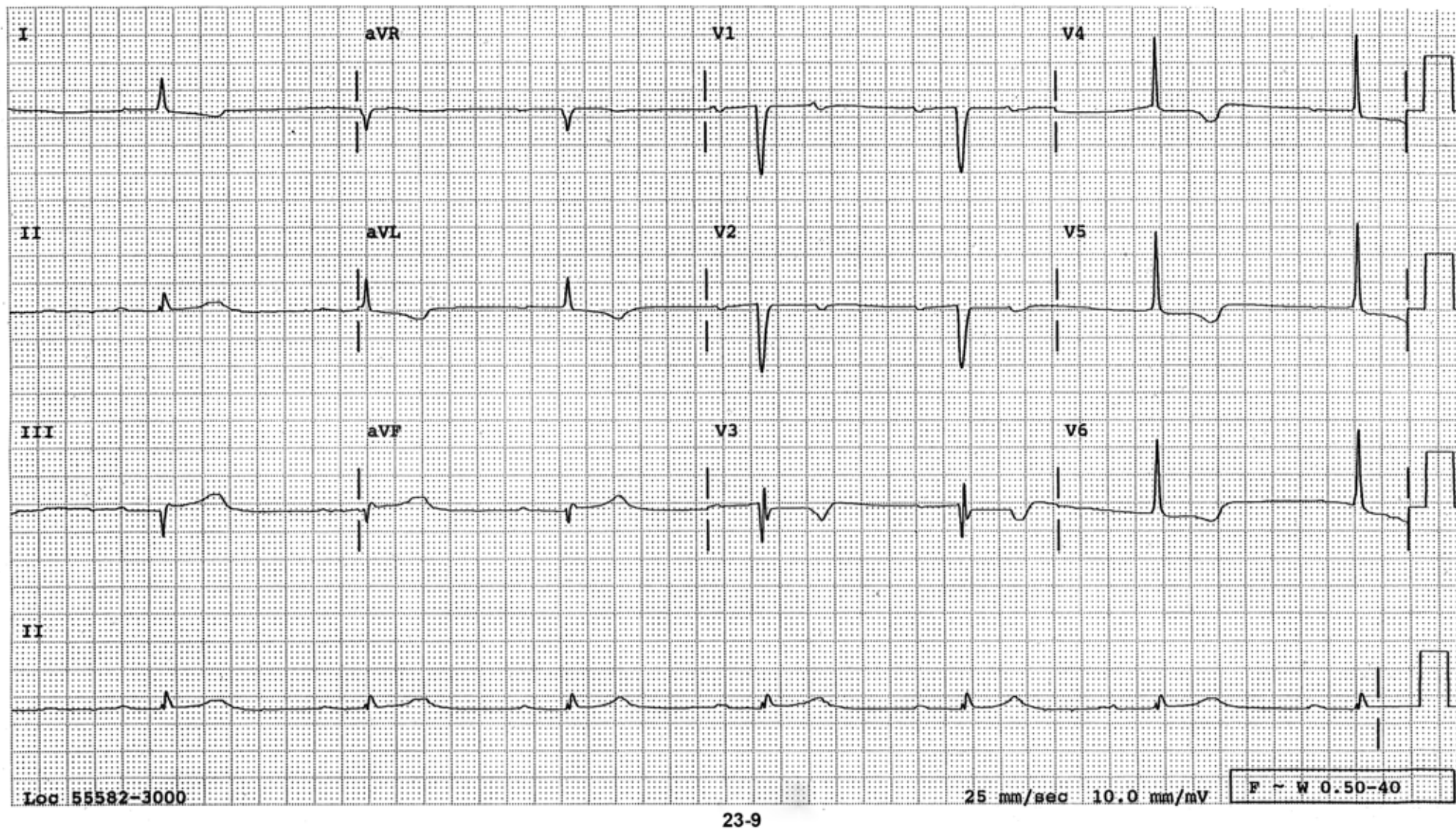
	<i>RBBB</i>	<i>Anterior Fascicular Block</i>	<i>Anterior MI</i>
(A)	Yes	Yes	Yes
(B)	Yes	Yes	No
(C)	No	Yes	Yes
(D)	Yes	No	Yes



Answer: (A) Yes, Yes, Yes

Discussion: Sinus tachycardia at 120/minute.

RBBB and left axis deviation reflect BIFB. Acute anteroapical STEMI is present.

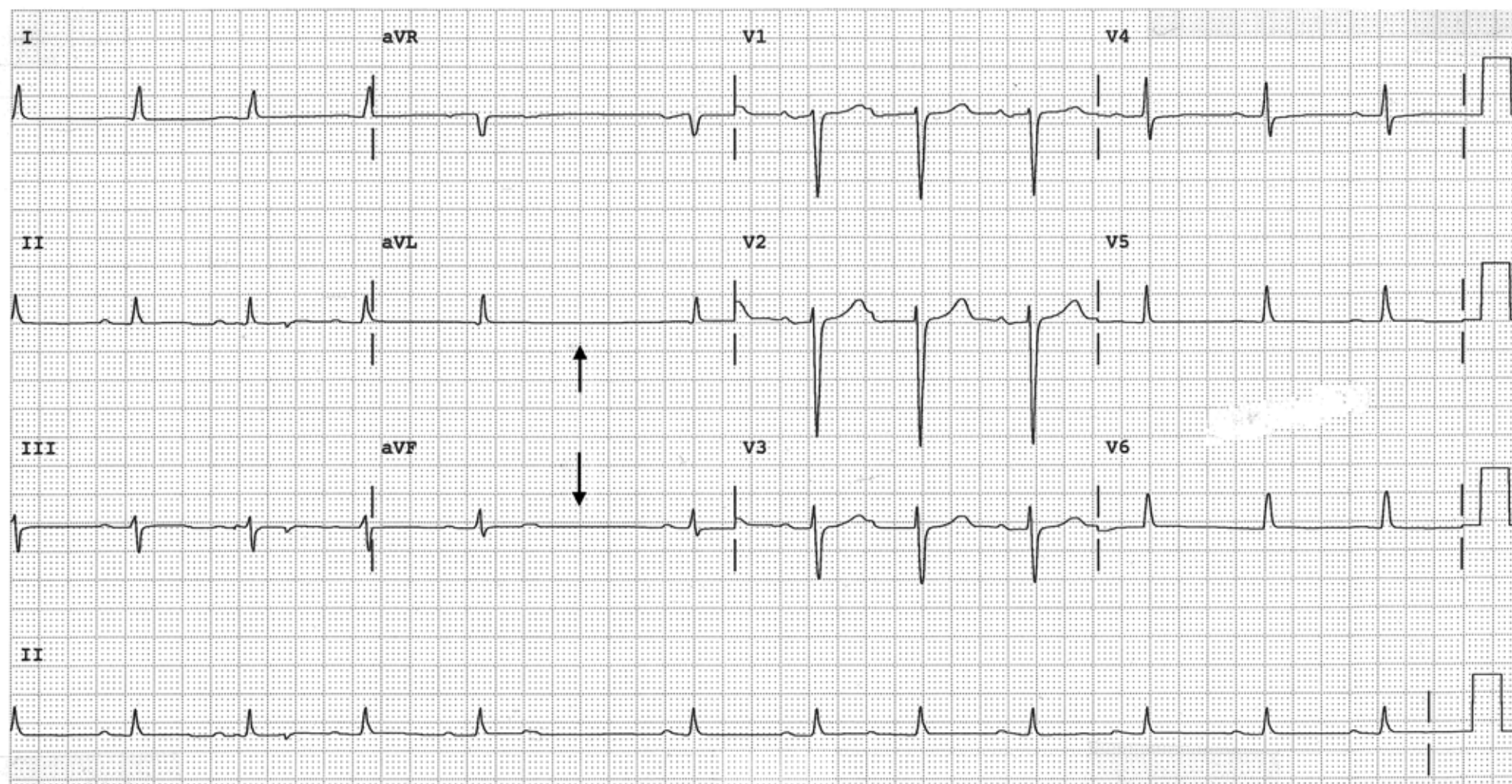


23-9 The tracing reveals NSR at a rate of 85/minute with 2:1 AV block. Q wave with ST elevation in lead III indicates inferior infarction of undetermined age. QS pattern in V₁ and V₂, with a prominent Q wave in V₃, suggest old antero-septal infarction as well.

- Dx:
1. Sinus rhythm with 2:1 AV block
 2. Inferior infarct, age undetermined
 3. Old antero-septal infarction

23-10 Question: What is the reason for the pause (arrow) in the middle of the tracing? Choose one from below:

- (A) Sinus arrest or SA block
- (B) Second degree AV block
- (C) Nonconducted atrial premature beat
- (D) Artifact from the machine malfunction



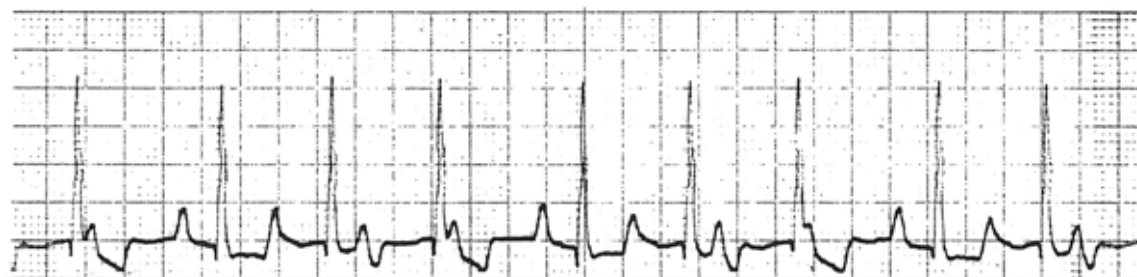
23-10

Answer: (C) Nonconducted atrial premature beat

Discussion: A blocked P wave is evident in the rhythm strip of lead II. This P wave occurs prematurely, not at regular interval, ruling out of 2° AV block.

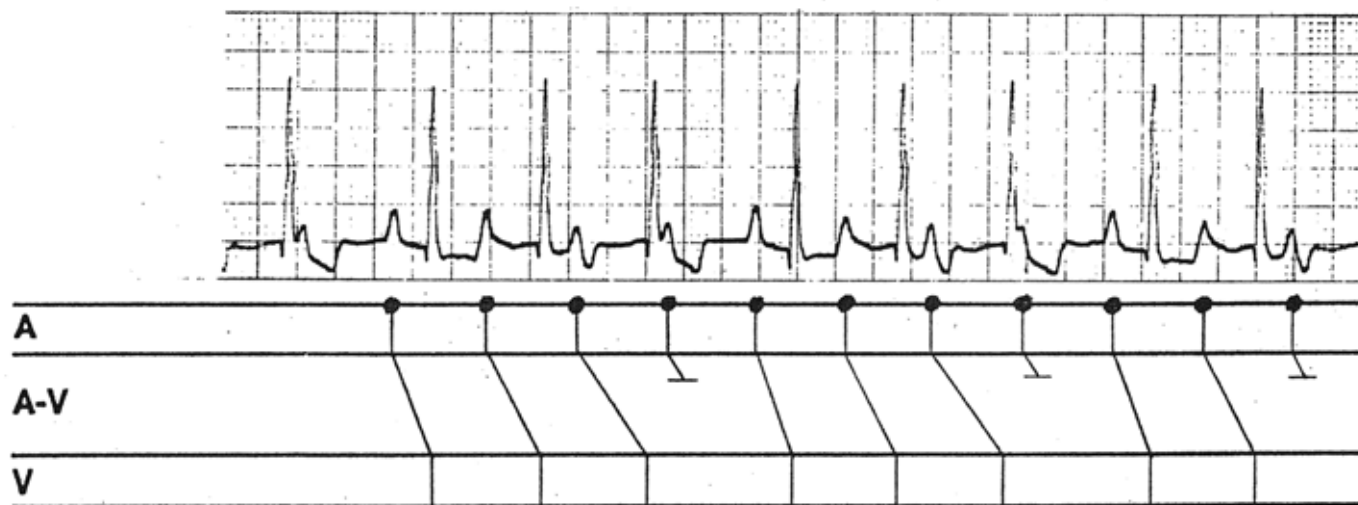
23-11 *Question:* This rhythm strip reveals (choose one from below):

- (A) MAT
- (B) Sinus tachycardia with AV Wenckebach phenomenon
- (C) Complete AV block
- (D) AV junctional tachycardia with AV dissociation



23-11A

Answer: (B) Sinus tachycardia with AV Wenckebach phenomenon as diagrammed in Figure 23-11B

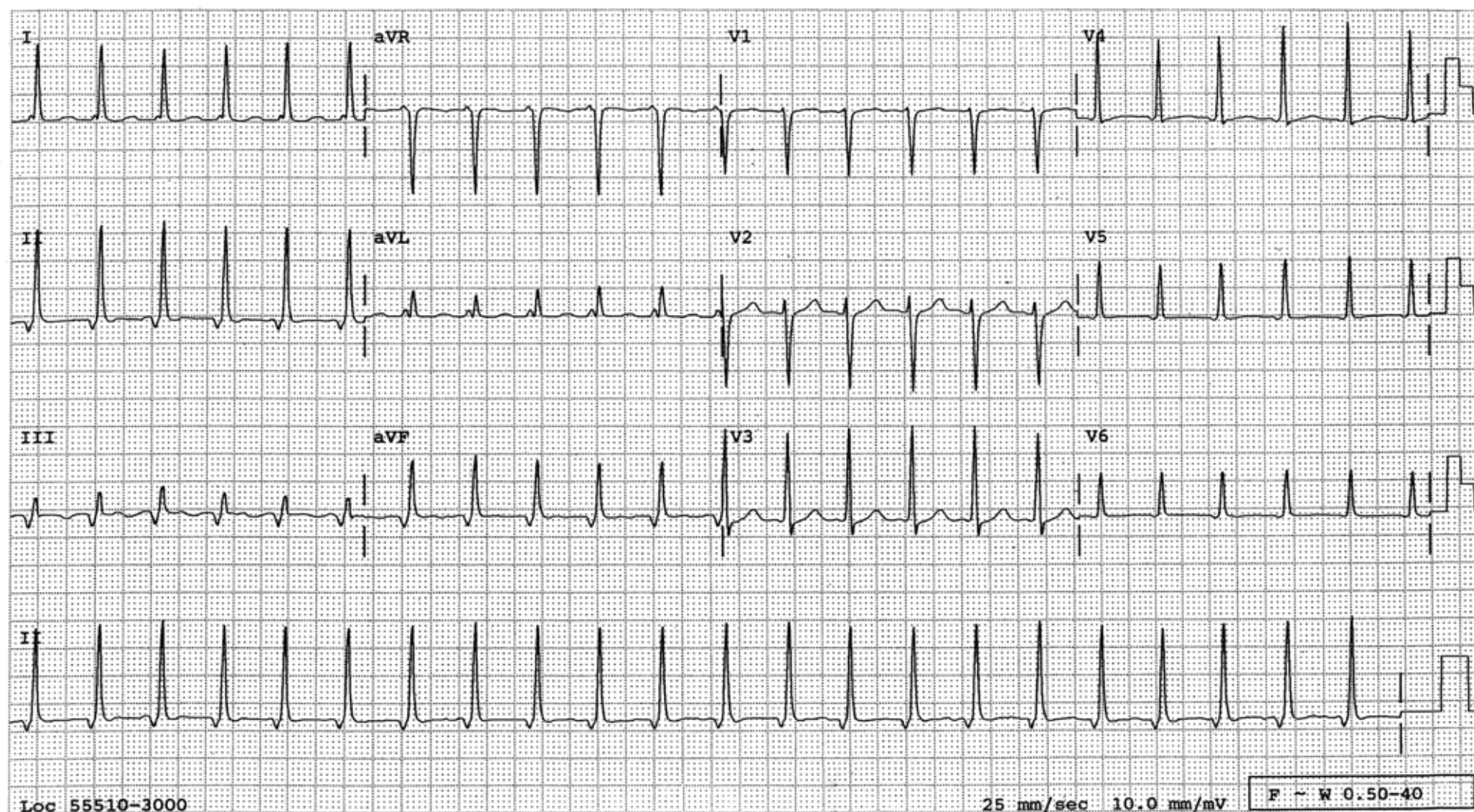


23-11B

Discussion: P waves occur regularly. P-R interval progressively lengthens until a P wave is blocked: typical of AV Wenckebach phenomenon. Regularly occurring P waves rule out MAT. In (C) and (D), QRSs should occur regularly, which is not the case here.

23-12 Question: This tracing reveals (choose one from below):

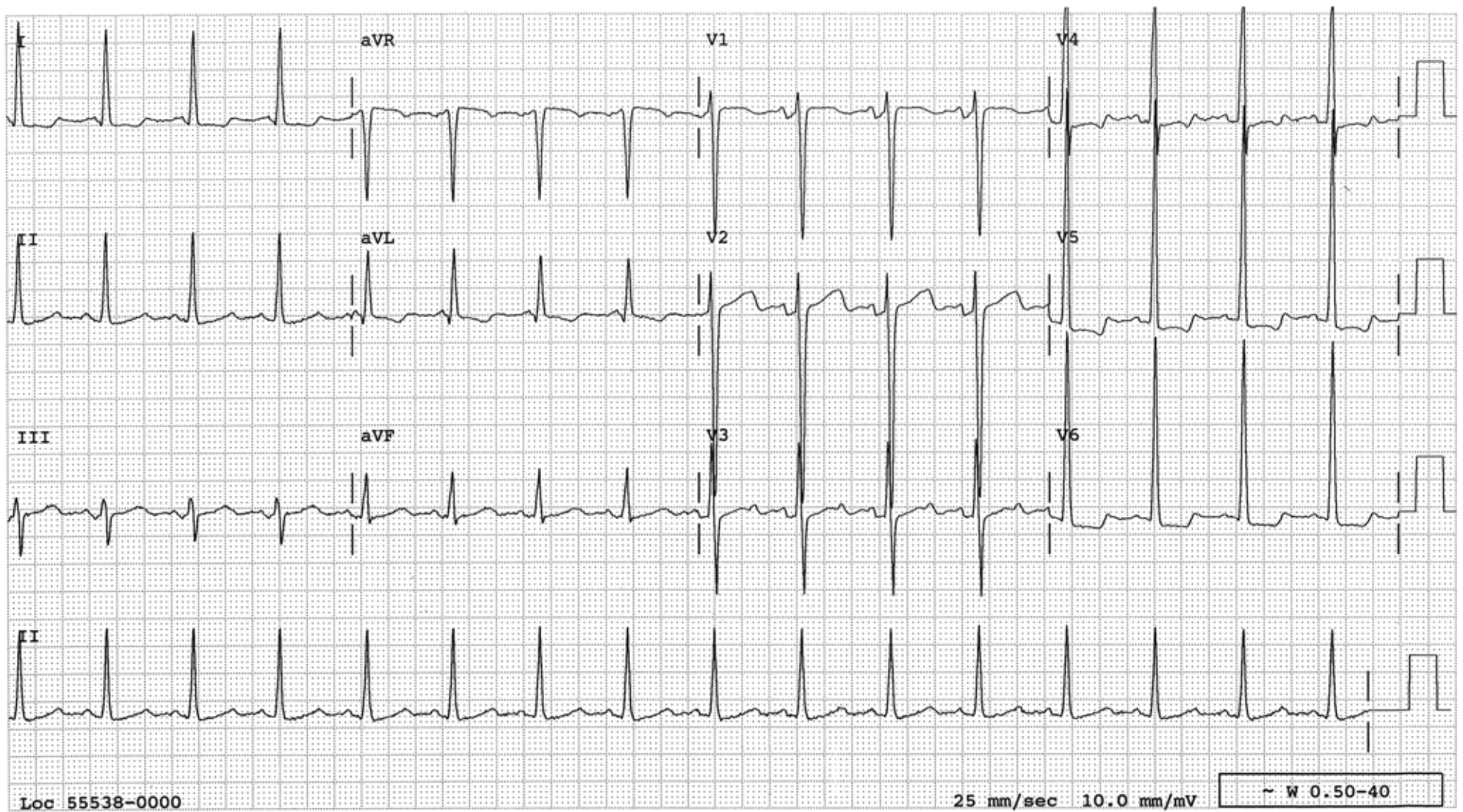
- (A) AV junctional tachycardia
- (B) Inferior MI
- (C) WPW syndrome simulating inferior MI pattern
- (D) Slow atrial flutter with 1:1 AV conduction



Answer: (A) AV junctional tachycardia

Discussion: Narrow QRSs occur regularly at a rate of 140/minute. The negative deflection in the inferior leads is a retrograde P wave just in front of junctional beats, not Q wave of an infarction or inverted delta waves when timed with QRS in other leads.

SECTION 24

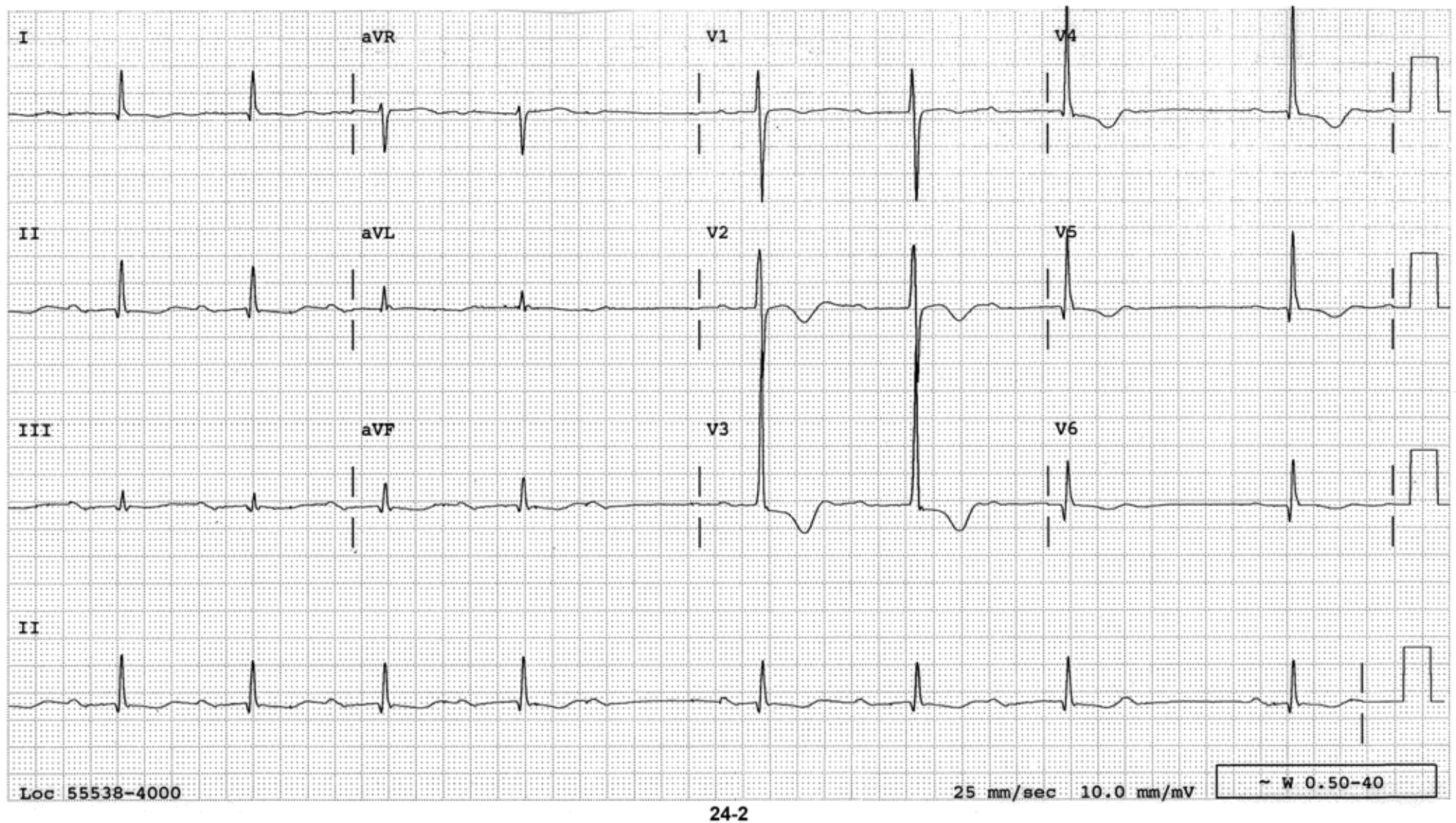


24-1

24-1 Normal sinus rhythm at a rate of 94 minutes. Voltage criteria and ST-T changes for LVH are present.

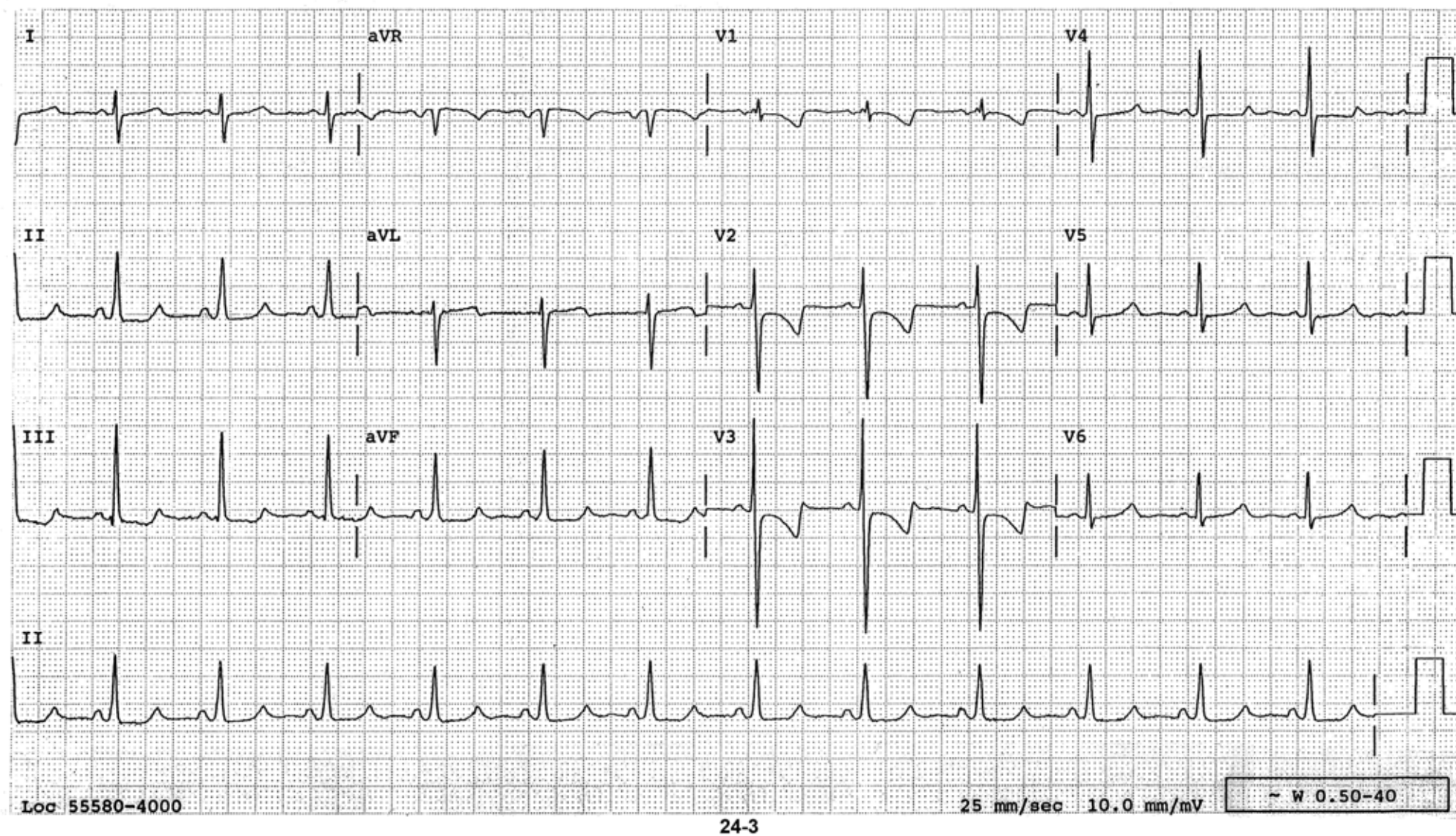
Dx: 1. NSR

2. LVH



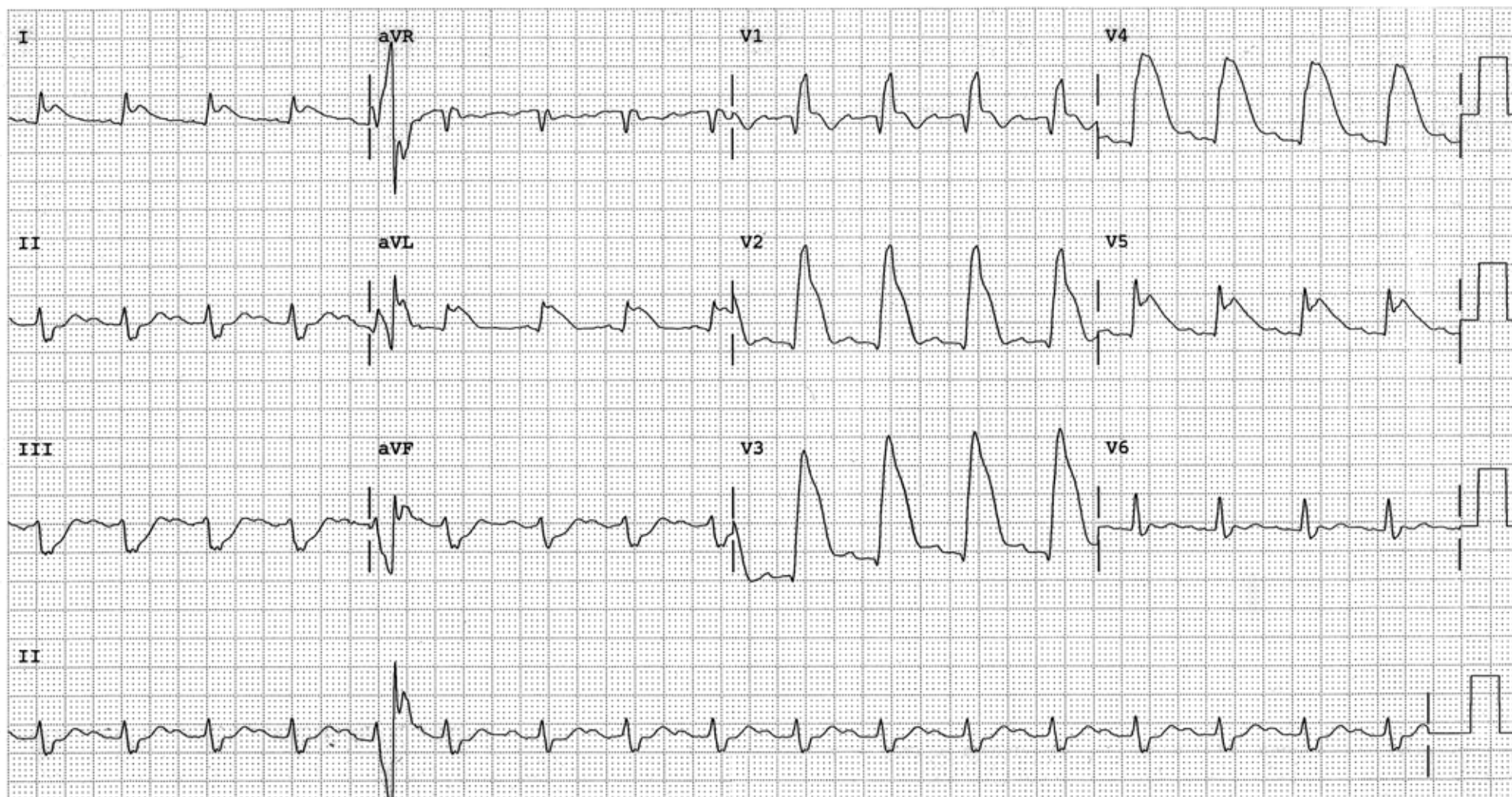
24-2 P waves occur regularly at a rate of 60/minute. The P-R interval progressively lengthens until eventually a P wave is blocked; a typical AV Wenckebach phenomenon. Small but broad Q waves in lead III, combined with tall R waves in the right precordial leads and T wave inversion most likely reflect inferoposterior MI, which may be responsible for this AV block.

- Dx:
1. NSR with Mobitz Type I 2° AV block
 2. Inferoposterior infarct



24-3 Normal sinus rhythm at a rate of ~80/minute. The QRS axis is shifted to the right. That, combined with a tall R wave in V_1 with a strain pattern ST depression in the right precordial leads is consistent with RVH.

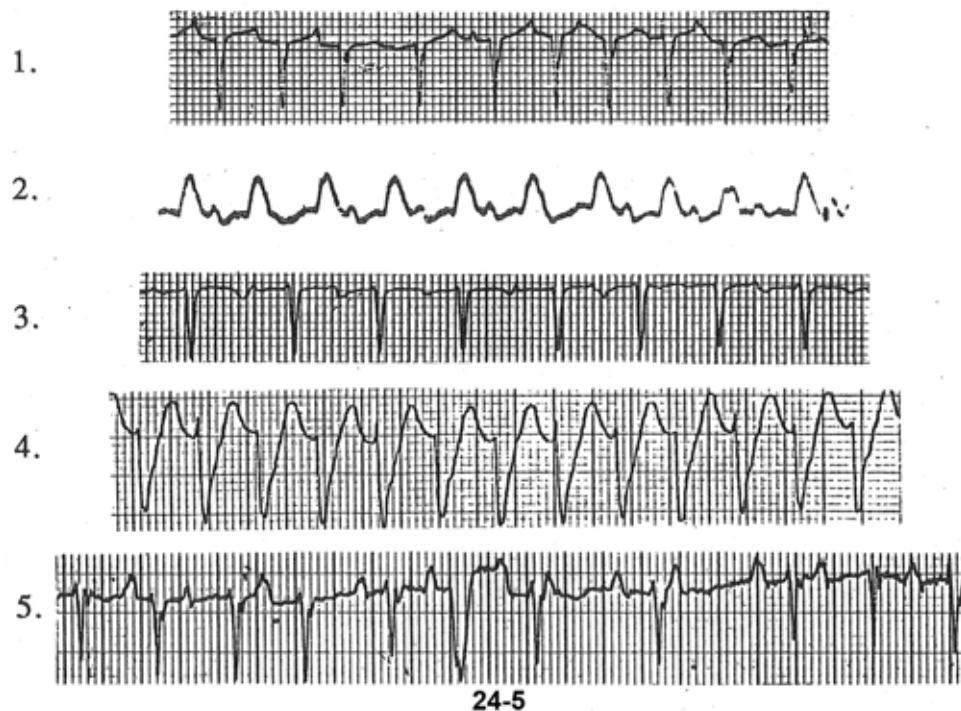
- Dx: 1. NSR
2. RVH



24-4

24-4 A sinus rhythm at a rate of 100/minute. The P-R interval is prolonged to 240 milliseconds. A QR pattern in V_1 with a marked ST elevation in other precordial leads, as well as in leads I and aVL, indicate acute anterior MI with RBBB. ST depression in lead III is a reciprocal change of the findings seen in aVL. Marked ST elevation, as seen in V_3 and V_4 of this tracing, can be mistaken for widened QRS.

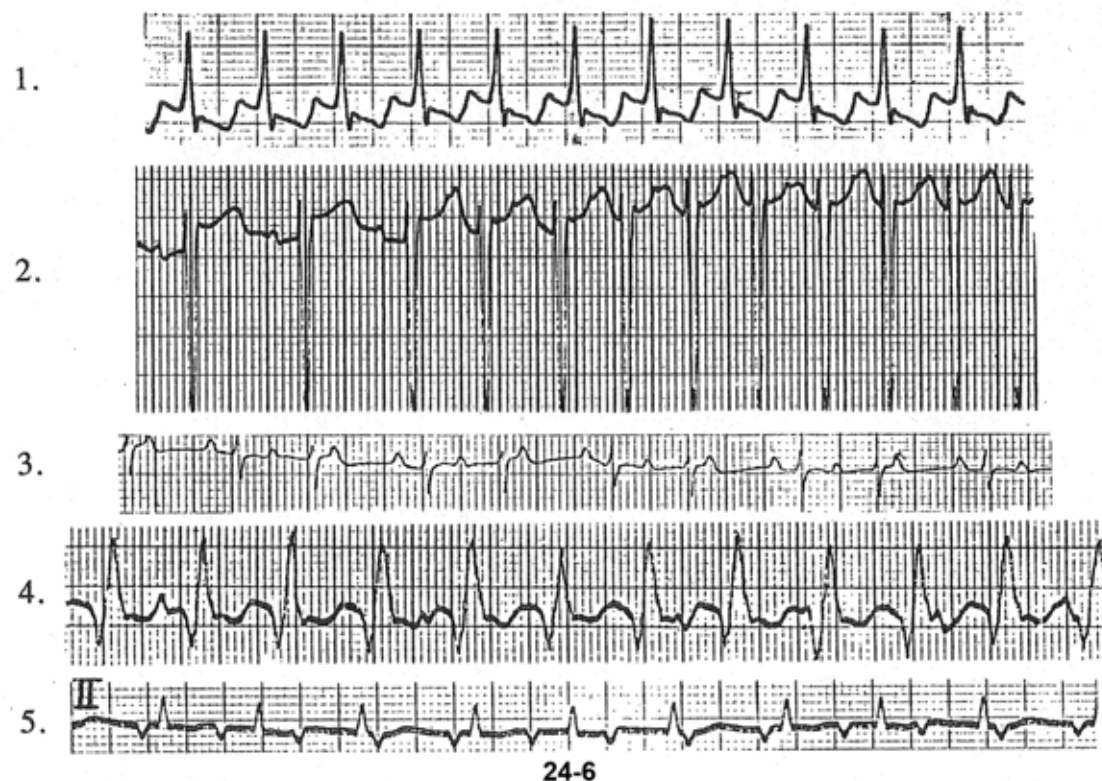
- Dx:
1. NSR
 2. Acute anterior MI
 3. RBBB



24-5 *Tracings 1, 3 and 5:* MAT—P waves occur irregularly at a rate of more than 110/minute with changing morphology. These features are diagnostic of MAT.

Tracing 2: It reveals wide QRS tachycardia at a rate of 150/minute. There are blips consistent with a P wave after some QRSs but not with others. Besides, the QRS-to-blip distance lengthens until finally a QRS is not followed by a blip. These features are characteristic of VT with retrograde VA Wenckebach phenomenon (4:3 conduction ratio in the middle of the strip).

Tracing 4: VT—Wide QRS tachycardia at a rate of about 170/minute. This is a proven case of VT.



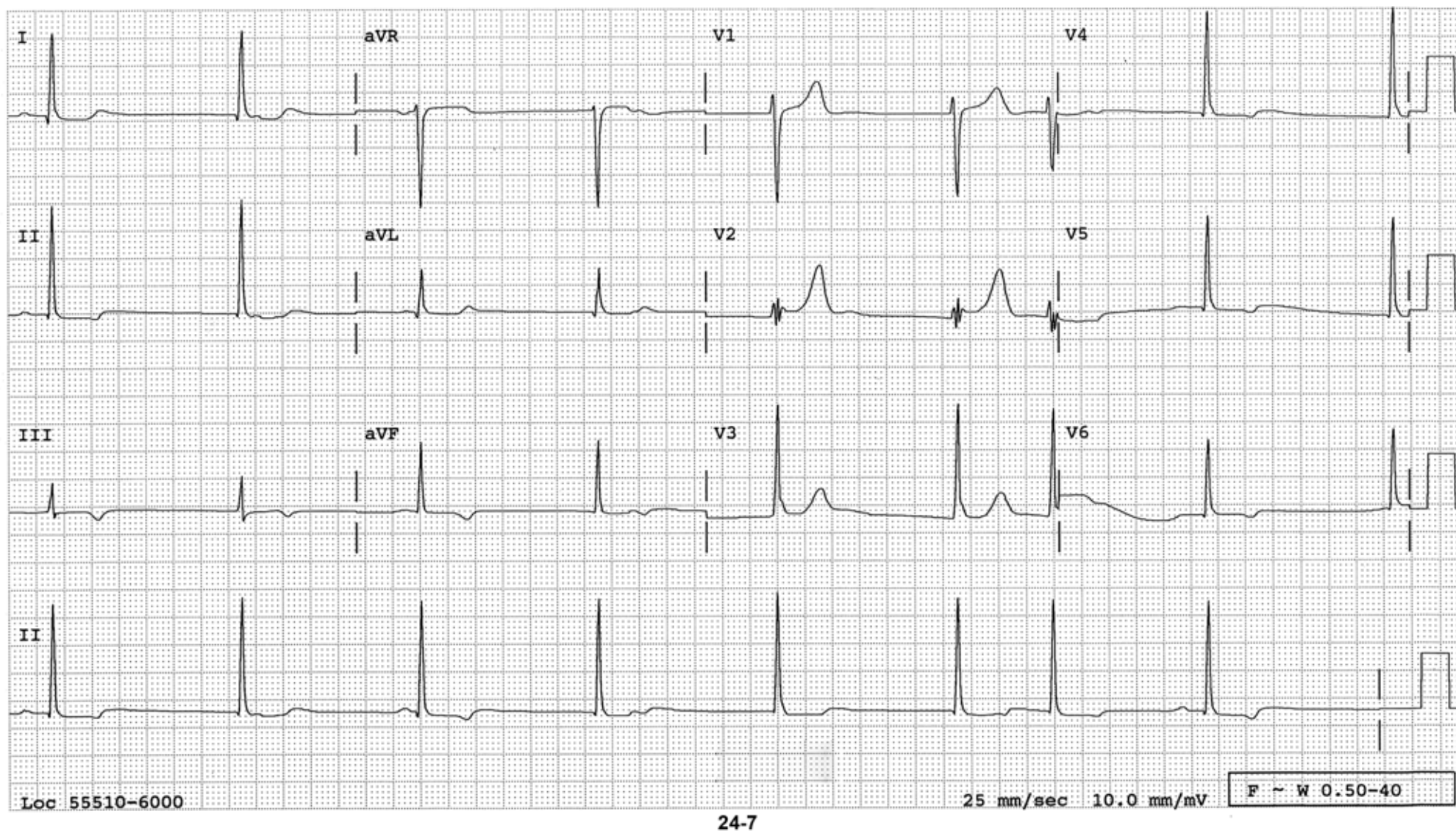
24-6 *Tracing 1:* This is a proven case of atrial flutter with 2:1 AV conduction. The second flutter wave is lower than the one in front of the QRS due to the summation of ST depression.

Tracing 2: The fourth QRS is an atrial premature beat, which initiates a run of atrial tachycardia

Tracing 3: Atrial tachycardia at a rate of 170/minute is present. Every third P wave is blocked, and the preceding two P waves are conducted to the ventricles with a short-then-long P-R interval, a typical of 3:2 AV Wenckebach phenomenon. The 3:2 AV conduction ratio results in the QRSs being paired together as in this case. Digitalis toxicity should be strongly considered.

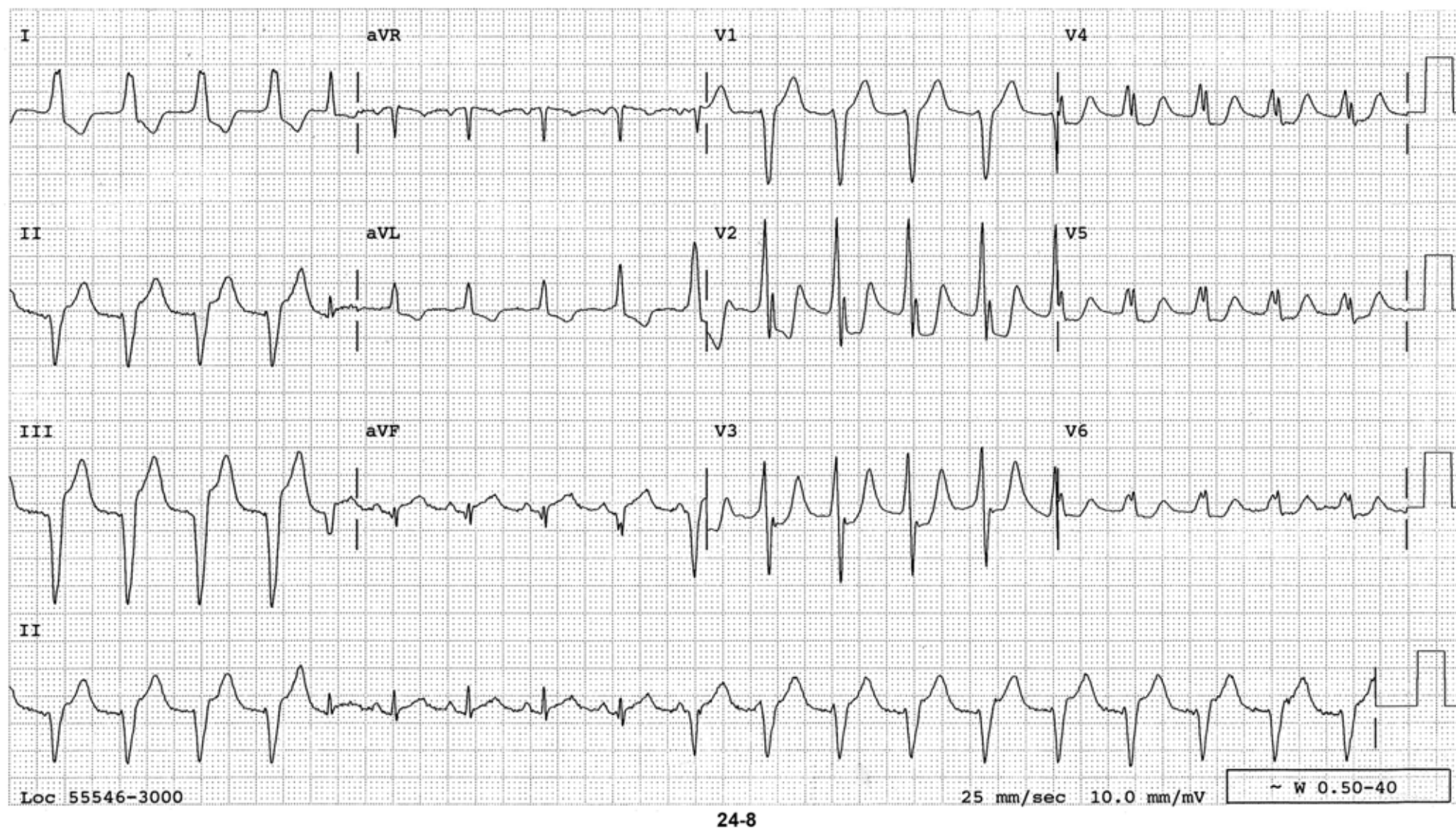
Tracing 4: A wide QRS tachycardia at a rate of 135/minute. P waves are seen occasionally which bear no relationship to the QRS. Two consecutive P waves are seen at the end of the tracing revealing the intrinsic sinus rate and other P waves occur at that P-P interval. This is an example of VT with complete AV dissociation.

Tracing 5: This is a rhythm strip of lead II. Small negative blips occur regularly at a rate of 150/minute, reflecting retrogradely conducted P waves. The P wave to the QRS is short initially, longer with the second beat, even longer with the third beat, and the fourth P wave is blocked. This cycle repeats again. This is either an AV junctional tachycardia or low atrial tachycardia with 4:3 JV or AV Wenckebach phenomenon. This tracing is from a patient with digitalis toxicity.



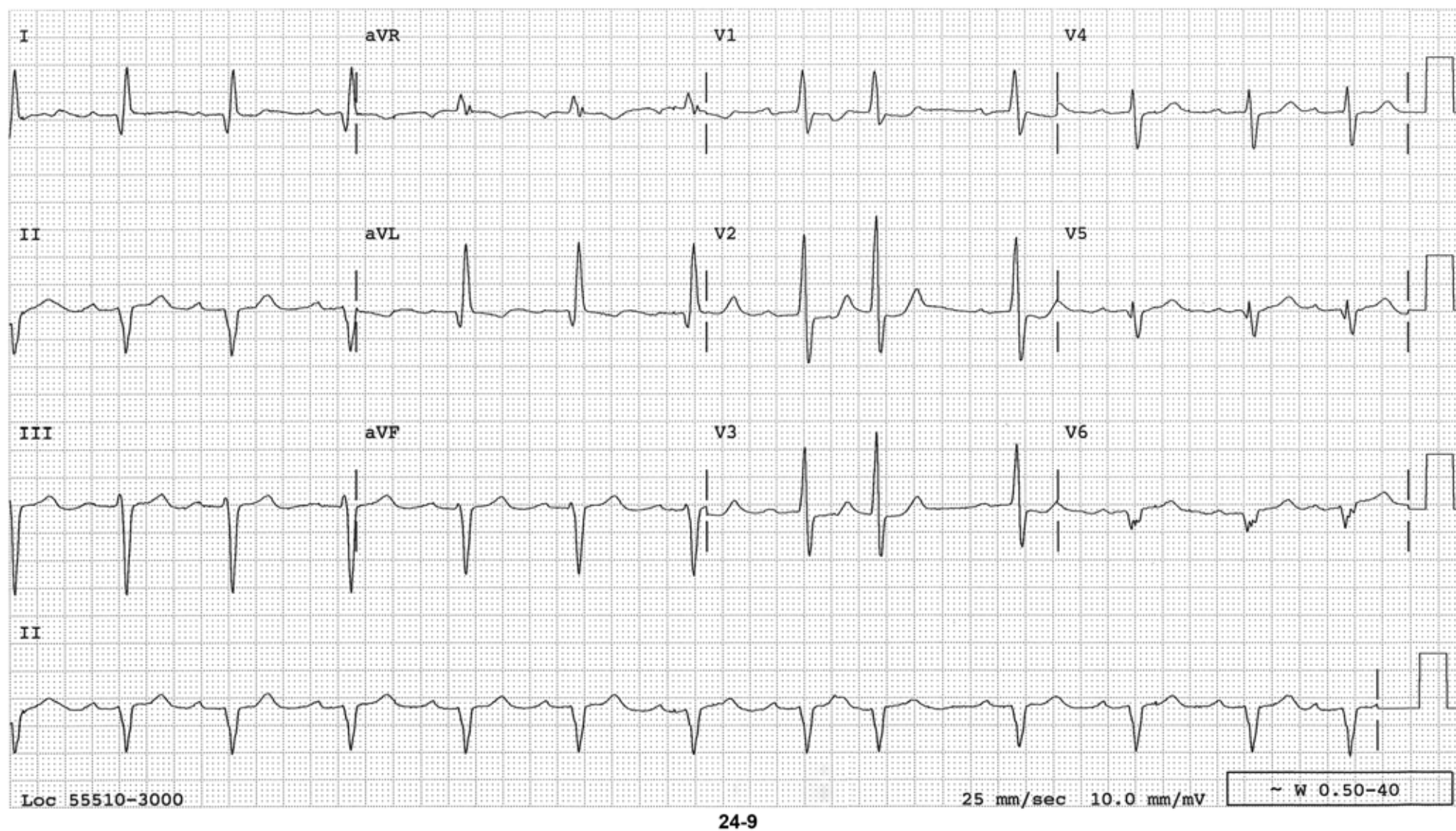
24-7 The sinus rate is very slow, occurring at about 35/minute, and this allows AV junctional rhythm to escape. The second beat from the end is an echo (reciprocating) beat and the last beat is a capture beat (the R-R interval is shorter than that of junctional rhythm). The voltage criteria and ST-T changes for LVH are present.

- Dx: 1. Sinus brachycardia, resulting in junctional escape rhythm with one echo beat and one capture beat
2. LVH



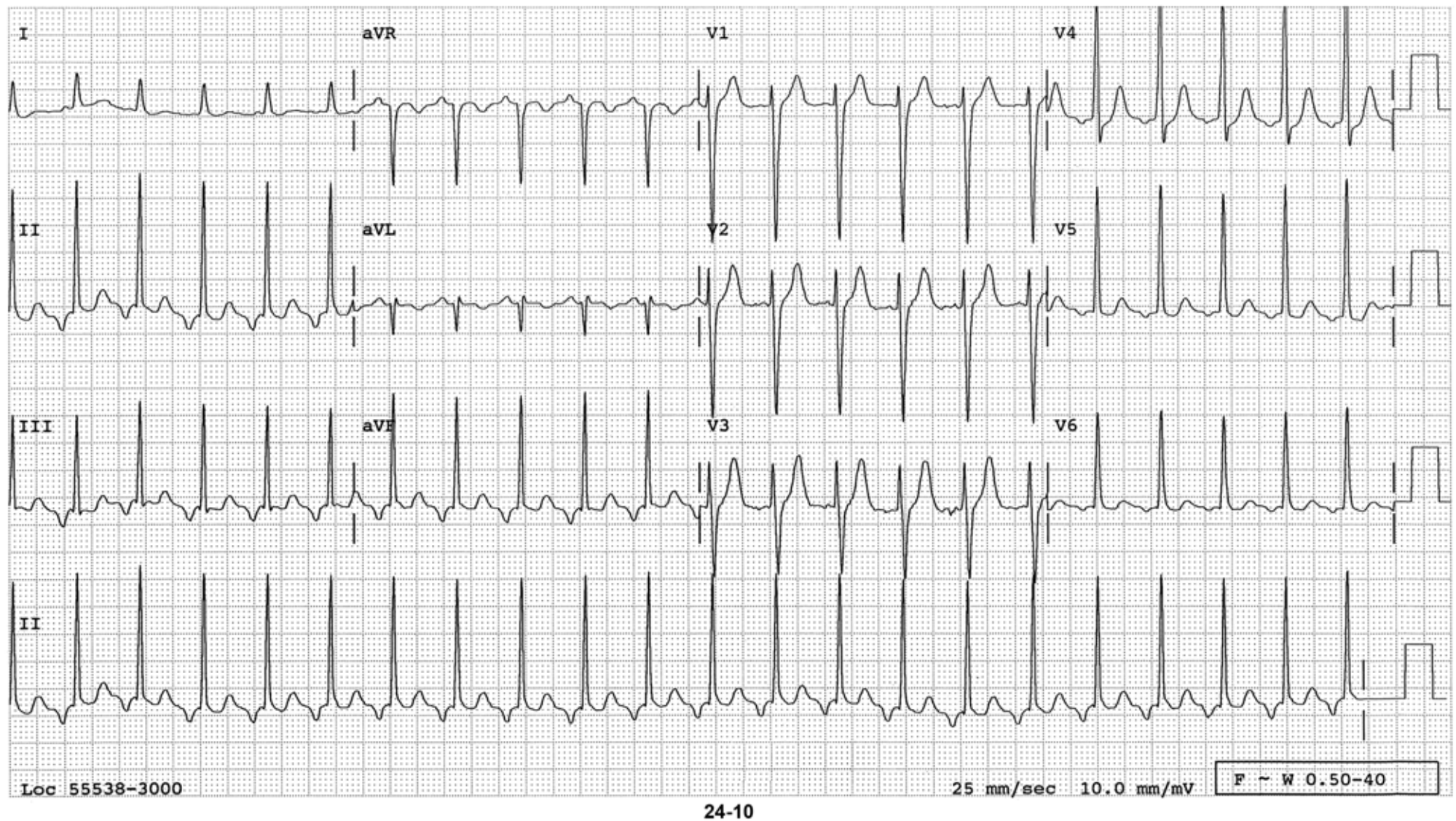
24-8 Wide QRS tachycardia is interposed with sinus rhythm. At the beginning of the second run of the wide QRS tachycardia, P waves are seen marching into the QRS, proving that this is a ventricular rhythm. The ventricular rate of 115/minute makes this an accelerated idioventricular rhythm. Only one normally conducted QRS complex is seen in lead III, and that complex has the findings of inferior infarct, which is the cause of this accelerated idioventricular rhythm. Posterior wall is also involved judging from the marked horizontal ST depression in V_2 .

- Dx:*
1. Sinus tachycardia
 2. Accelerated idioventricular rhythm 2° to acute inferoposterior infarct



24-9 Normal sinus rhythm at a rate of 70/minute. The P-R interval is prolonged to 250 milliseconds. Abnormal left axis deviation is consistent with LAFB. The R waves are tall in the right precordial leads which regress in the left precordial leads, and eventually it becomes a QS pattern in V₆. Q waves are wide and deep enough in leads I and aVL. These findings are consistent with posterolateral infarction. One PAC is present.

- Dx:*
1. NSR
 2. 1° AV block
 3. Atrial premature complex
 4. LAFB
 5. Posterolateral infarct

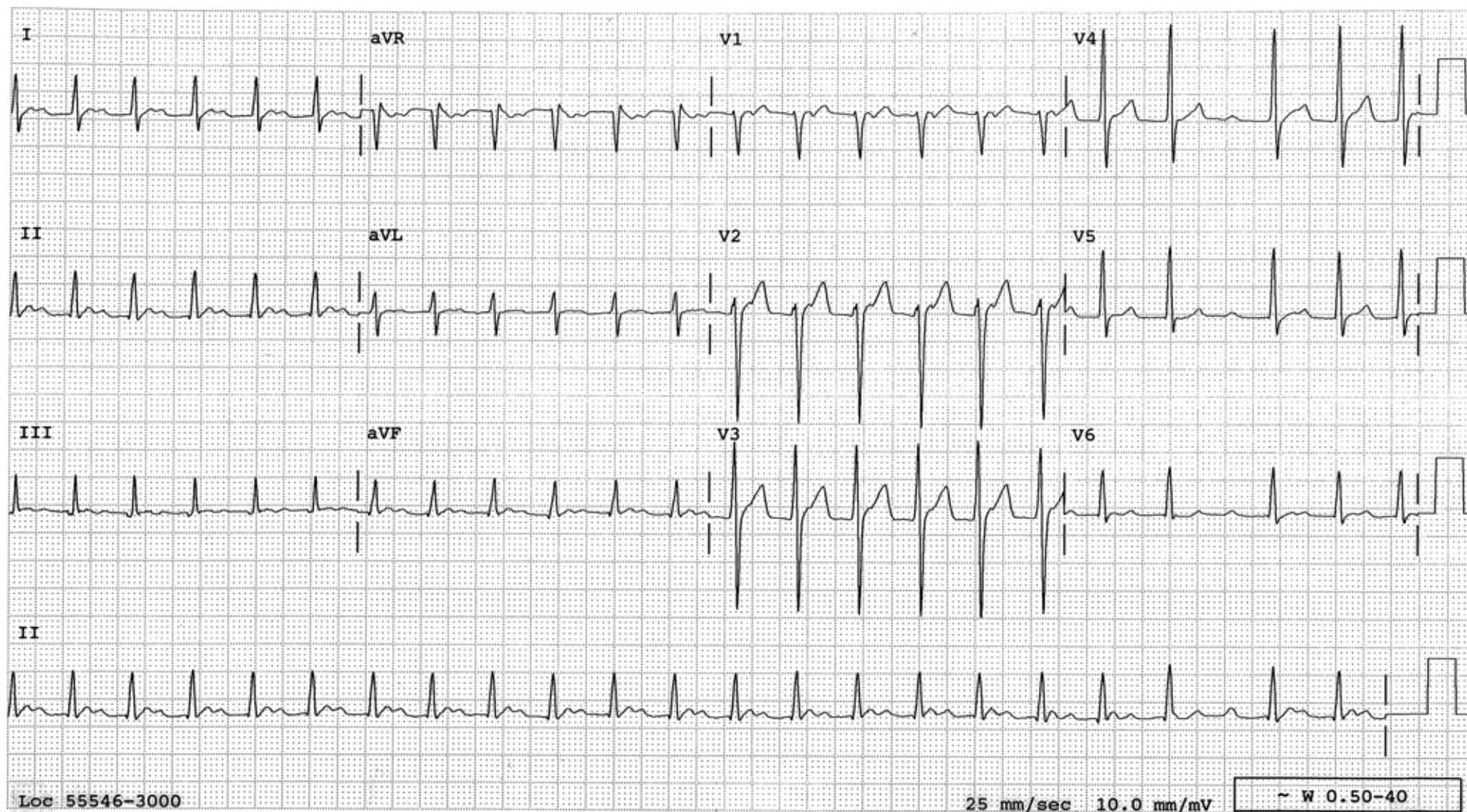


24-10 Narrow QRS tachycardia at a rate of 129/minute. The P waves are inverted in the inferior leads, suggesting either junctional rhythm or low atrial rhythm. The P-R interval of 120 milliseconds favors junctional tachycardia. Voltage criteria and ST-T changes for LVH are present.

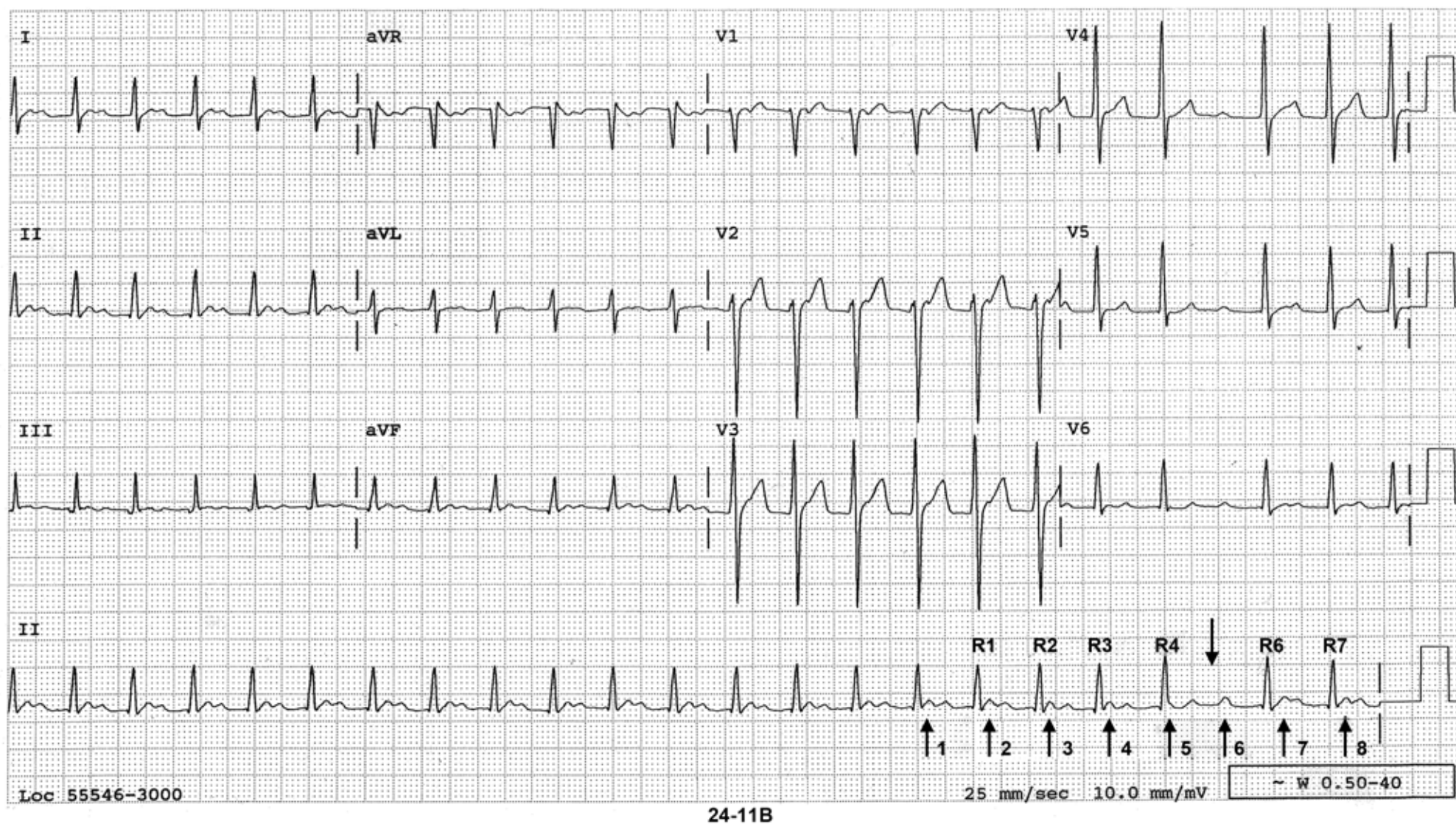
- Dx:*
1. Probable junctional tachycardia
 2. LVH

24-11 Question: This tracing reveals (choose one from below):

- (A) AV junctional tachycardia
- (B) Sinus tachycardia with Type I 2° AV block
- (C) Atrial flutter
- (D) Paroxysmal atrial tachycardia (PAT)



24-11A



Answer: (B) Sinus tachycardia with Type I 2° AV block

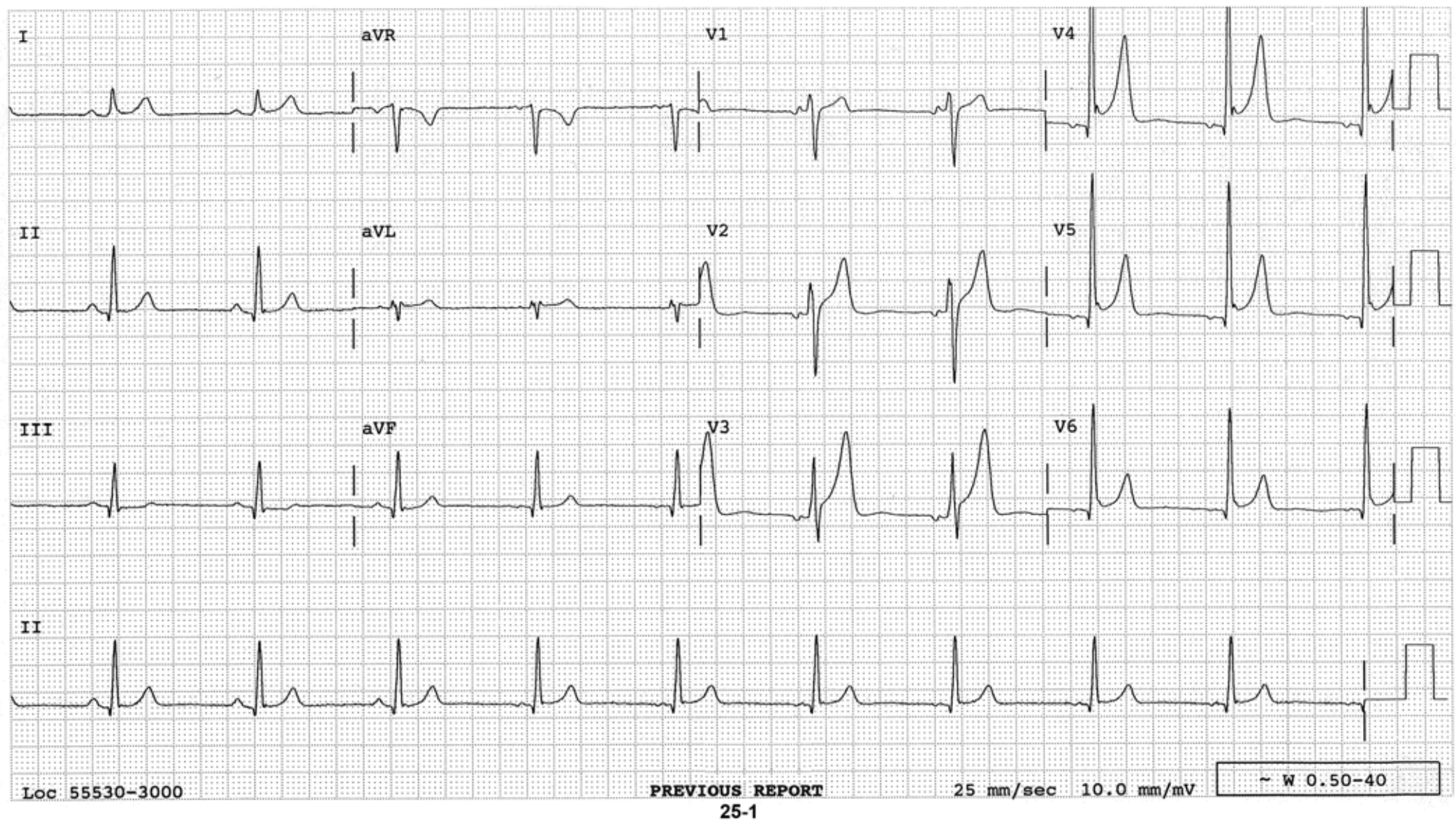
Clues: In the middle of the pause (↓) toward the end of the tracing, there is a deflection standing alone, and it cannot be anything but a P wave (↑₆). Similar deflections are occurring regularly at a rate of 140/minute (↑₁ through ↑₈) establishing a sinus tachycardia. ↑₁ through ↑₄ are not T waves since they peak too early from the preceding QRS. ↑₁-R₁, ↑₂-R₂, and ↑₃-R₃ intervals are constant, but ↑₄-R₄ interval is definitely longer and ↑₅ is blocked. ↑₆-R₆ interval is shortest and ↑₇-R₇ interval is longer. Thus, a new Wenckebach cycle begins again. When there is a regular rhythm and the P-QRS relationship is difficult to sort out, a pause which breaks the regularity may reveal crucial information as it did in this case.

ECG recording showing a premature ventricular contraction (PVC) in lead II. The PVC is labeled "C" with an arrow pointing to it. The recording shows leads II, A, A-V, and V. The PVC is a wide, bizarre QRS complex that occurs early and is not preceded by a P wave. The rhythm is regular sinus.

Primary disorder: Junctional acceleration. *Secondary phenomenon:* AV dissociation with occasional capture complexes (“c”)

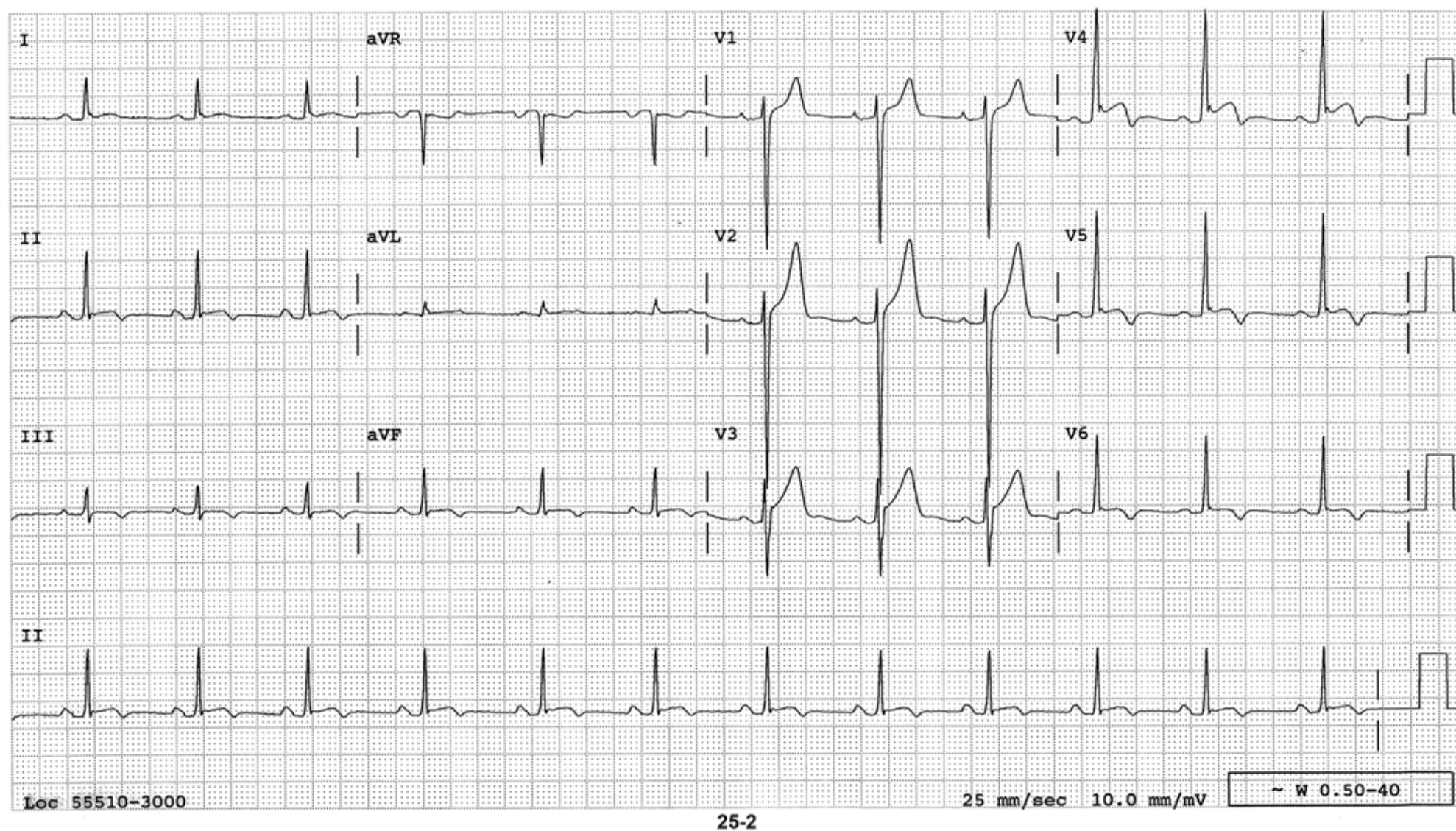
One should look for digitalis toxicity, myocardial ischemia or infarction or sympathetic overactivity, which are the common causes of accelerated junctional rhythm. The AV dissociation in this case is due to physiologic refractoriness of the conduction system or myocardium. The capture complexes are useful in proving that there is no AV block. Whenever the P wave occurs outside of the refractory period, the impulse is conducted to the ventricles resulting in a capture beat. The hallmark of the capture beat is that the complex occurs with a shorter R-R interval than others. The fourth QRS (↓) is induced by the sinus impulse (note that the R-R interval is 60 milliseconds shorter than other R-R intervals with a reasonably placed P wave in front of it).

SECTION 25



25-1 Normal sinus rhythm at a rate of 59/minute. The ST-segment is elevated in most of the precordial leads. Notching in the junction, concavity upward and small ratio of ST:T amplitude are all characteristic of early repolarization pattern. In pericarditis the ST:T amplitude ratio is greater. Mild PR-segment depression is also not unusual for this early repolarization pattern. The T waves are tall in the precordial leads which also is not uncommon for early repolarization pattern. In fact, that is the reason why the ST:T amplitude ratio is small.

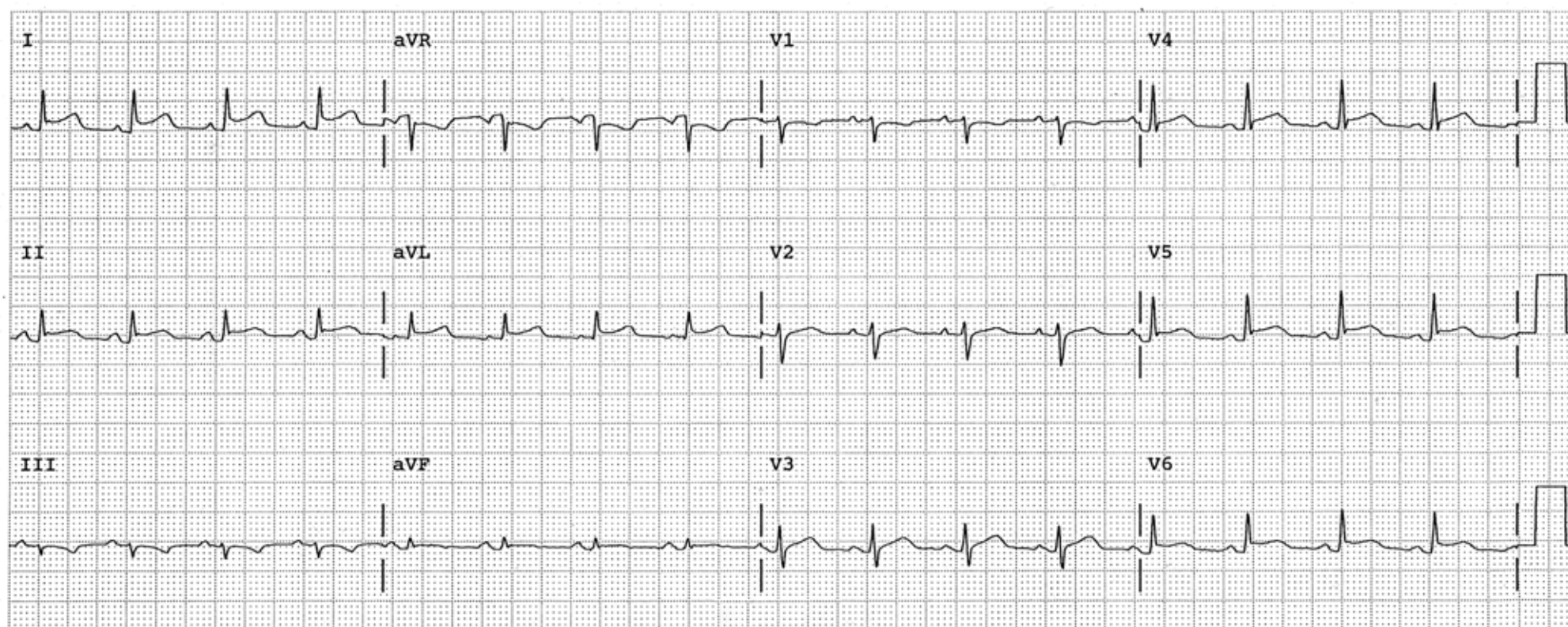
- Dx: 1. NSR
2. Early repolarization pattern as a normal variant



25-2 The ST segment is elevated diffusely with terminal T wave inversion. There is a notch at the junction in V_4 . The QT interval tends to be short. This is another form of normal variant seen almost exclusively in blacks and hardly in other races. This entity is still different from the early repolarization pattern in that the T waves are inverted while they are upright and tall in early repolarization. Unlike early repolarization, this entity is not widely recognized and is often mistaken to reflect an acute pericarditis or even STEMI. Some of these patients are taken to the angiography lab, only to find clean coronary arteries.

- Dx: 1. NSR
2. ST elevation of "the other" normal variant

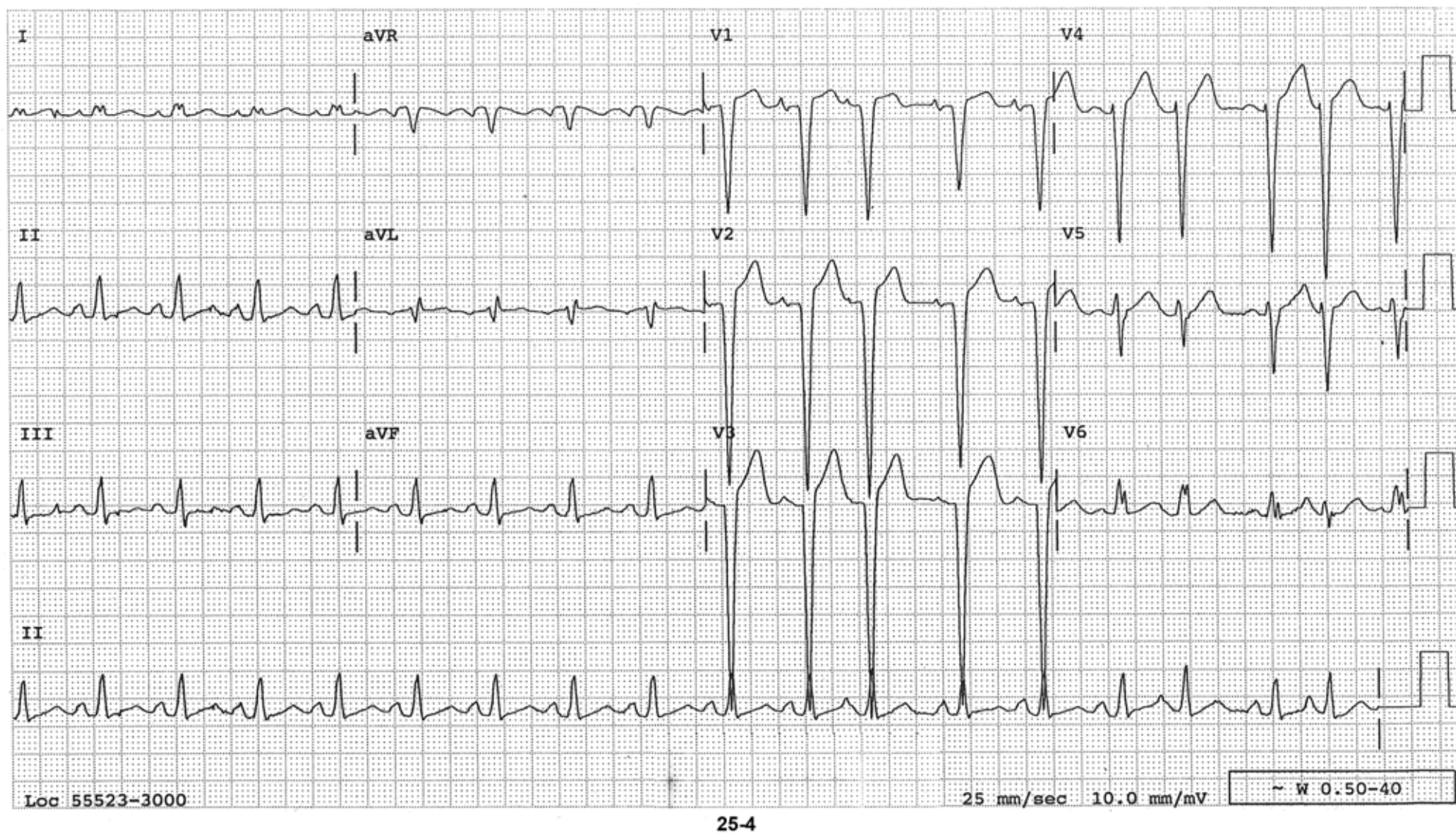
What clinical condition should this ECG tracing make you think of?



25-3

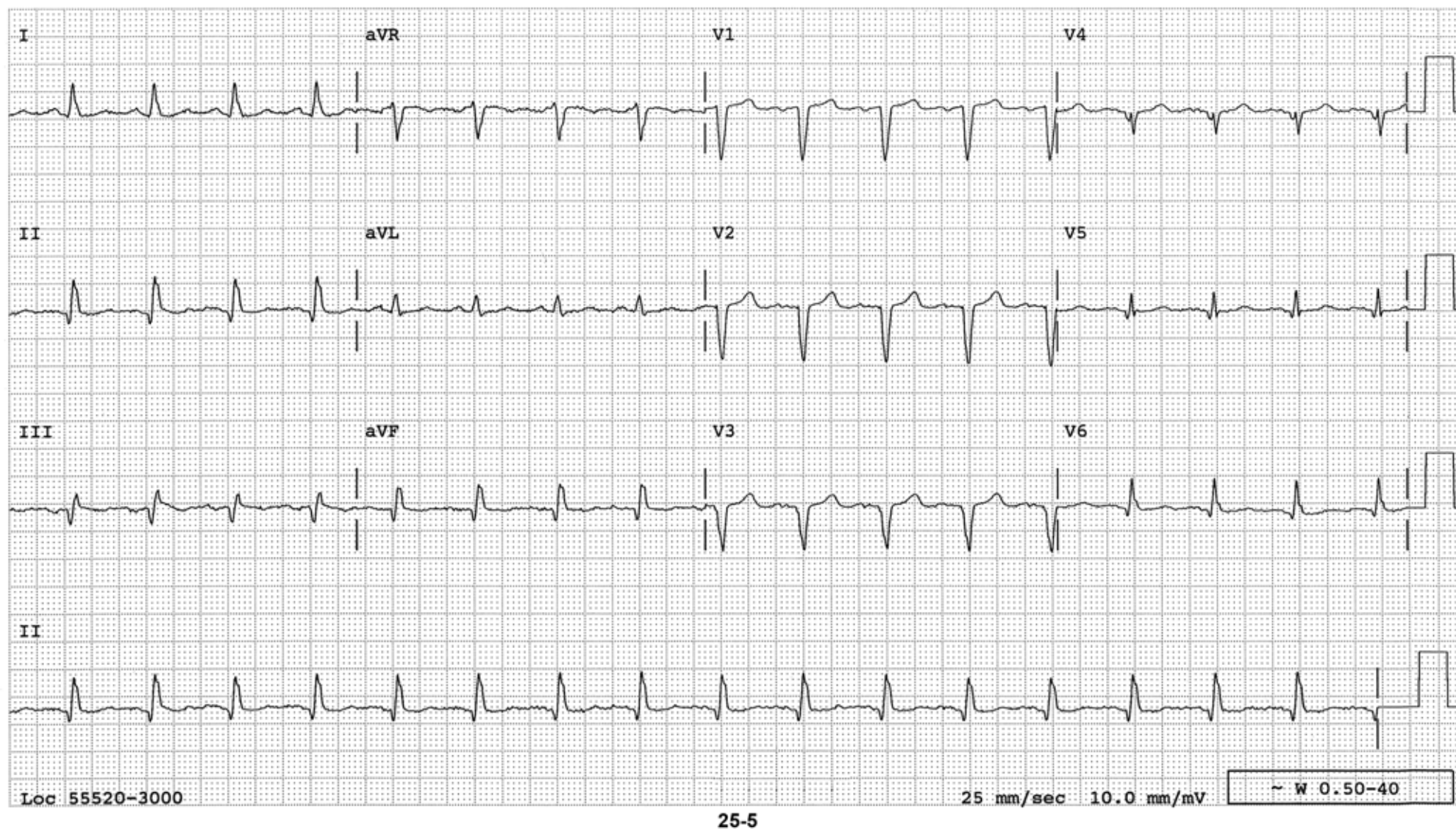
25-3 Normal sinus rhythm at a rate of 95/minute. The ST-segment is elevated and PR-segment is depressed diffusely. The lack of notching in the junction, especially in lead V₄, convex ST-segment, and high ST:T amplitude ratio favor pericarditis.

- Dx: 1. NSR
2. Acute pericarditis



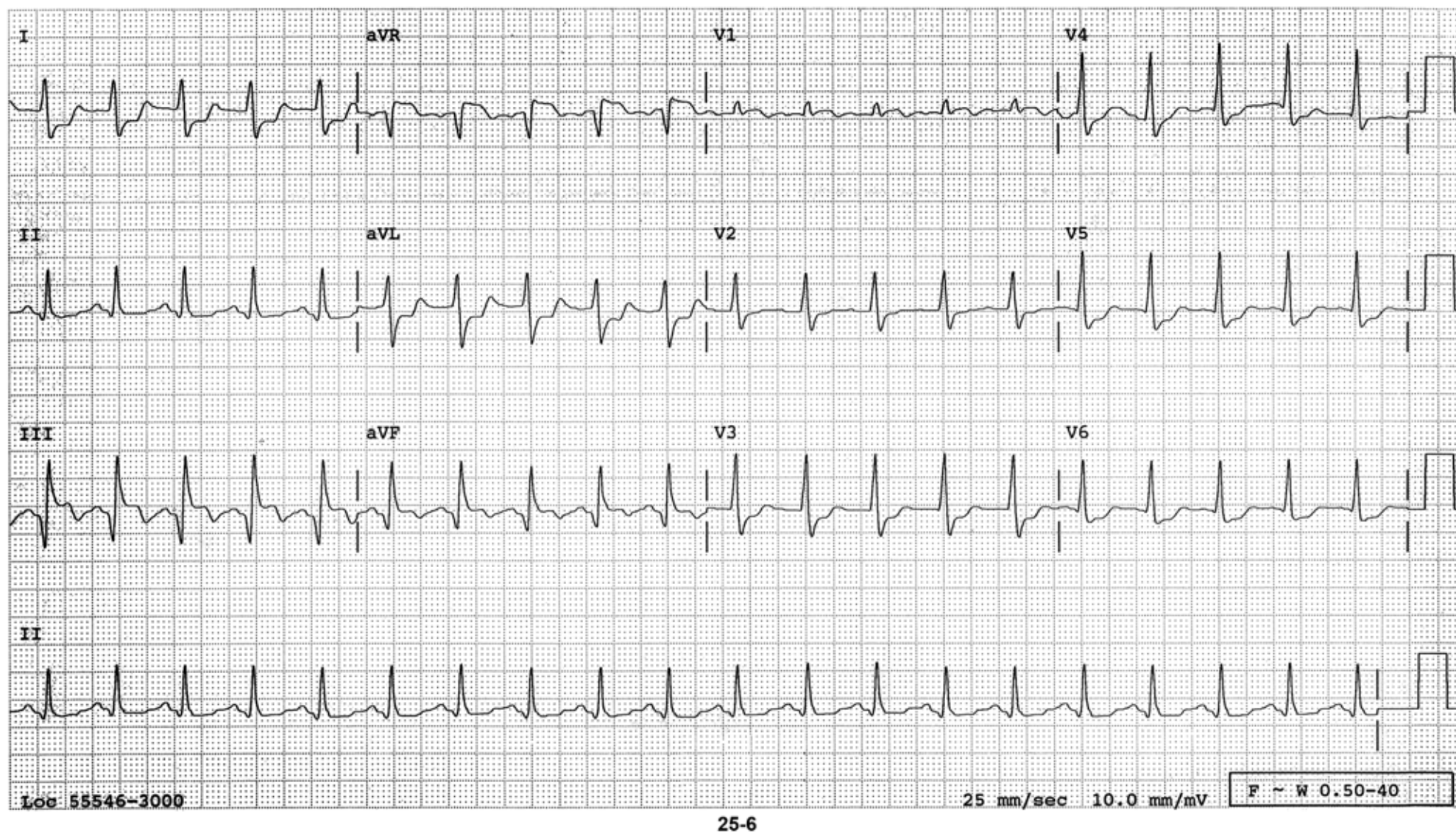
25-4 Sinus tachycardia at a rate of 110/minute with three PACs. QS pattern and ST elevation in V_1 - V_3 suggest acute anteroseptal infarction but they are all just from LVH. Note the ST-segment is concave. Acute infarction would more likely have caused a convex ST-segment. In fact, this kind of tracing is quite often mistaken for an anterior STEMI.

- Dx: 1. Sinus tachycardia with PACs
2. LVH



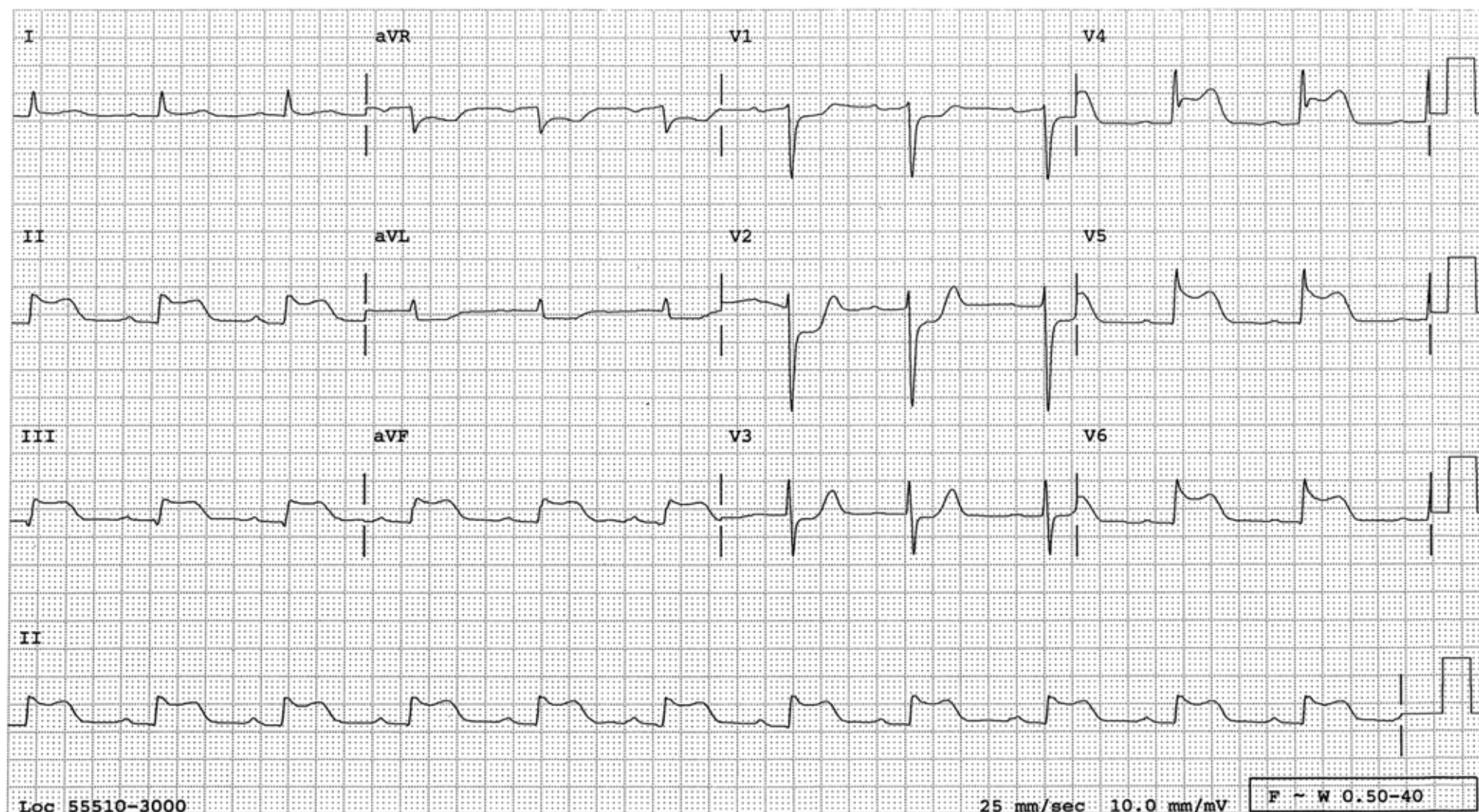
25-5 Normal sinus rhythm at a rate of 100/minute. The Q waves are wide and deep enough in the inferior leads, indicating old inferior MI. QS pattern extending to V₄ with a slightly prominent Q wave in V₅ also indicate old anterior MI.

- Dx:
1. NSR
 2. Old inferior infarct
 3. Extensive old anterior infarct



25-6 Sinus tachycardia at a rate of 120/minute. Prominent Q waves in the inferior leads with ST elevation and terminal T wave inversion, combined with tall R waves in the right precordial leads with horizontal ST depression in V_3 , reflect an acute inferior-posterior STEMI. ST-segment is slightly elevated in V_1 , indicating RV involvement, which is already predicted by the ST depression in lead I. In inferior MI, ST depression in lead I means the ST vector is pointed down and to the right. Why to the right? Because RV is involved.

- Dx:*
1. Sinus tachycardia
 2. Acute inferoposterior STEMI with RV involvement

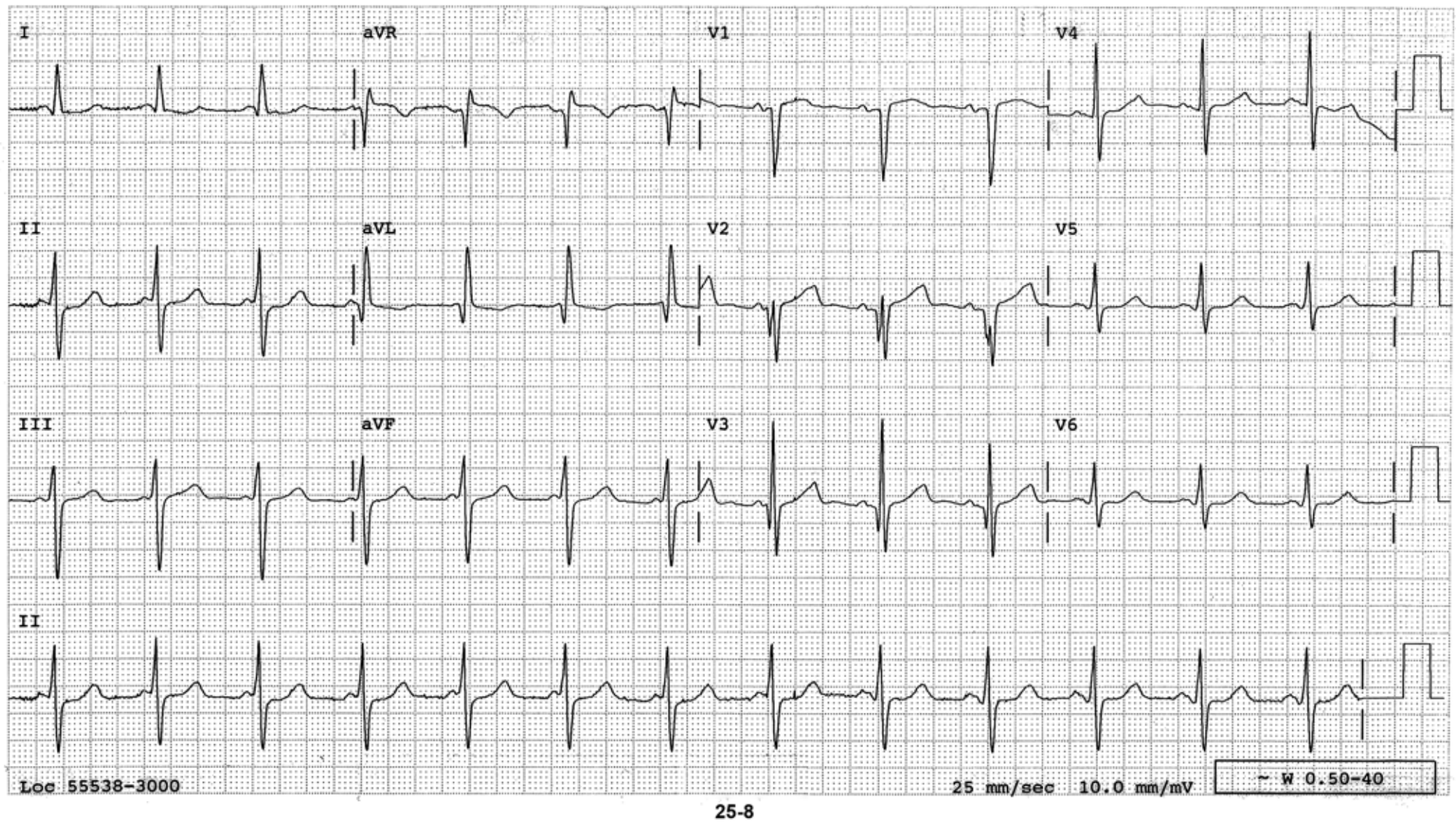


25-7

25-7 Sinus rhythm at 66/minute with 1° AV block.

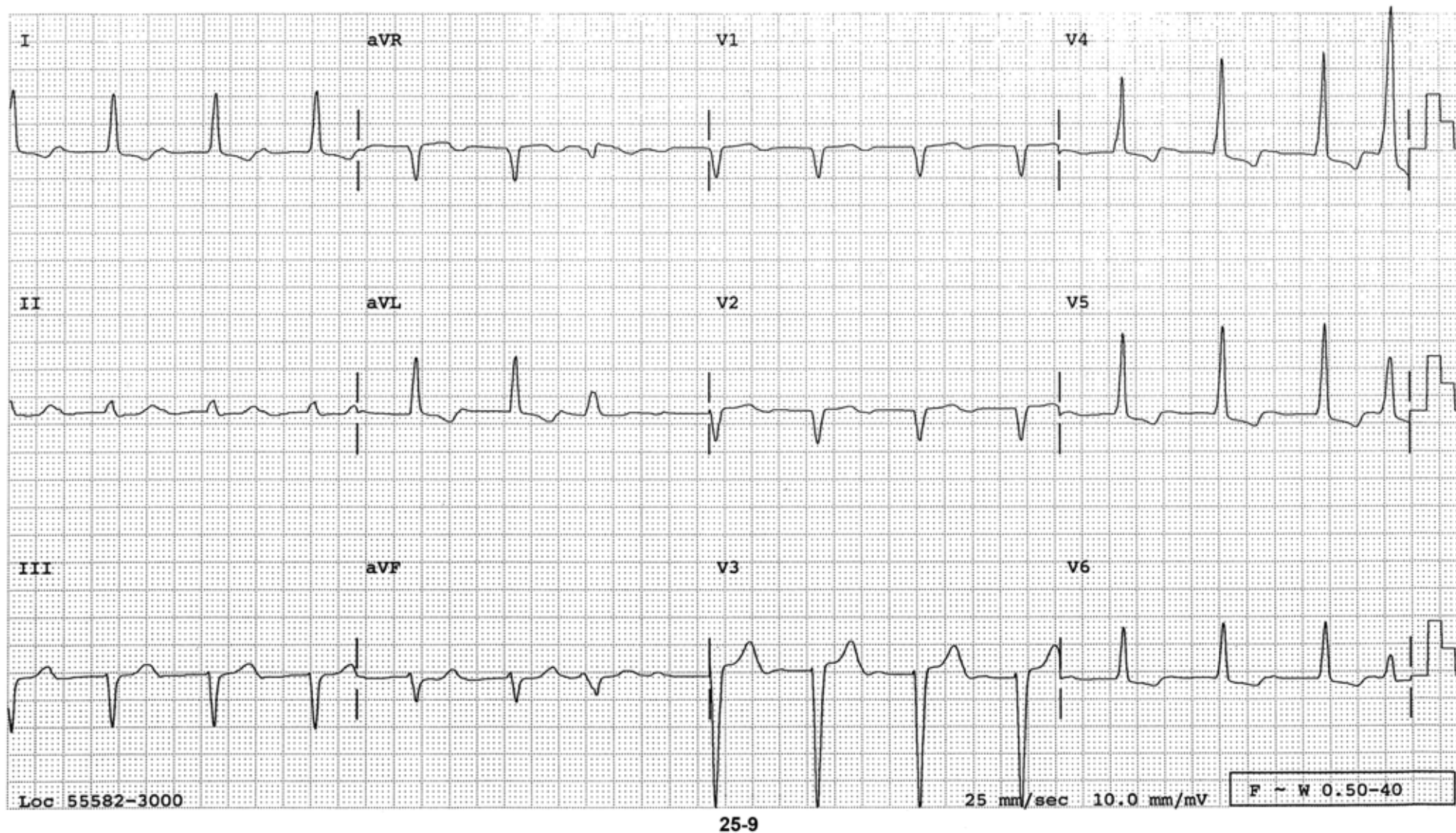
ST elevation in inferolateral leads with horizontal ST depression in V_1 - V_3 is diagnostic of STEMI of inferoposterolateral wall. ST is reciprocally depressed only in aVL, not in lead I indicating RV is not involved, and the culprit lesion must be not in proximal RCA but either RCA not proximal or circumflex coronary artery.

- Dx:
1. Sinus rhythm with 1° AV block
 2. STEMI of inferoposterolateral wall



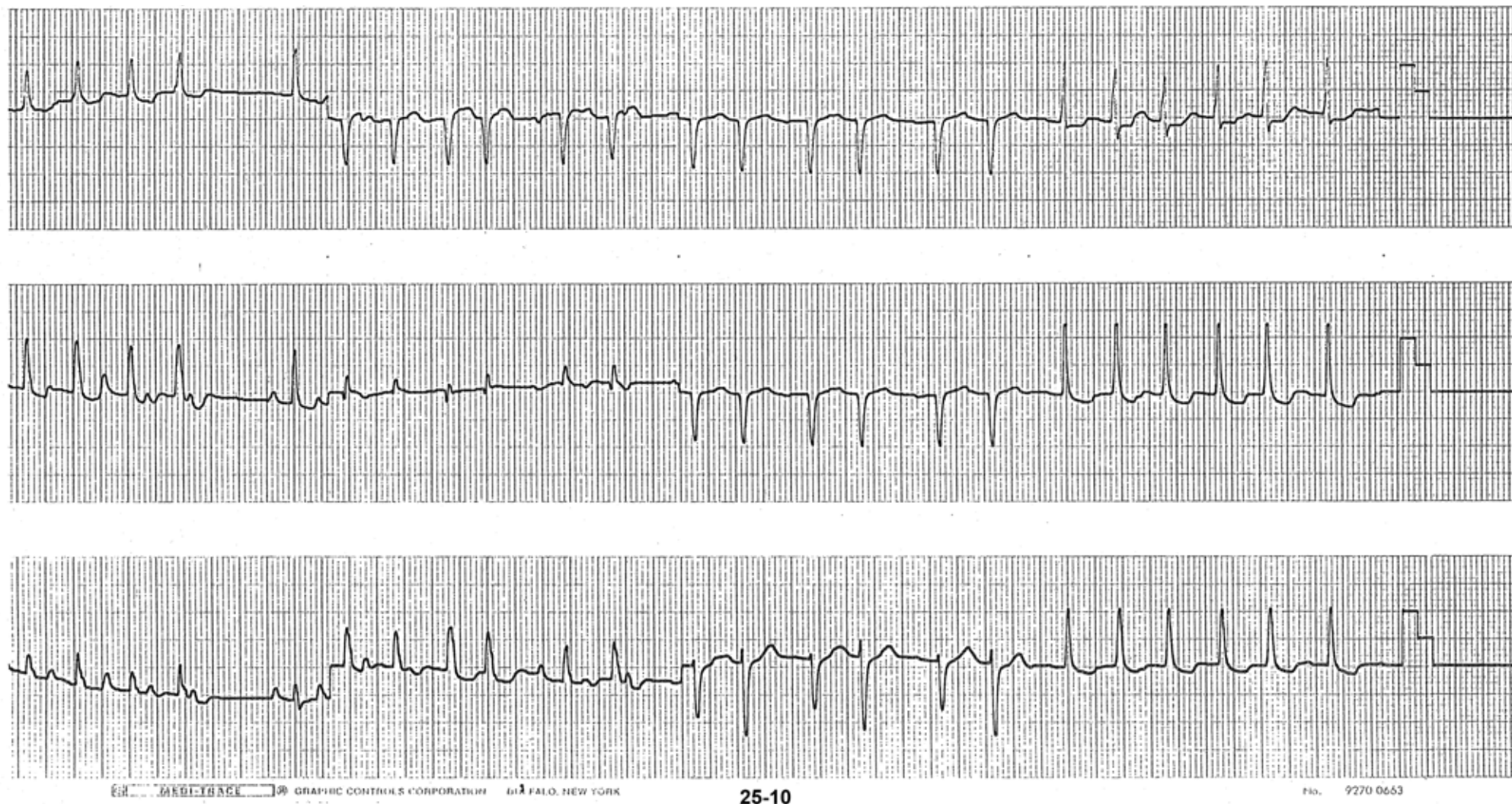
25-8 Regular rhythm at a rate of 80/minute. There is a P wave in front of each QRS, but the P-R interval is too short for it to have caused the QRS. Rather, AV junctional pacemaker happened to be accelerated to the rate similar to the sinus rate and AV junctional rhythm with AV dissociation results. QS pattern in V_1 , combined with a wide and deep enough Q wave in V_2 and V_3 indicates anteroseptal infarction. Myocardial ischemia or infarction is known to accelerate either junctional or idioventricular rhythm.

- Dx:*
1. Accelerated junctional rhythm with AV dissociation
 2. Anteroseptal infarct



25-9 Regular rhythm at a rate of 85/minute. The P wave is not easily recognizable. The compensatory pause following a PVC (the last complex of the limb leads) clearly reveals a P wave with a long P-R interval, documenting sinus rhythm with 1° AV block. The QRS voltage and ST-T changes in the precordial leads meet the criteria of LVH. QS pattern in V_1 and V_2 with a tiny R wave in V_3 could well be part of LVH rather than additional anteroseptal infarct.

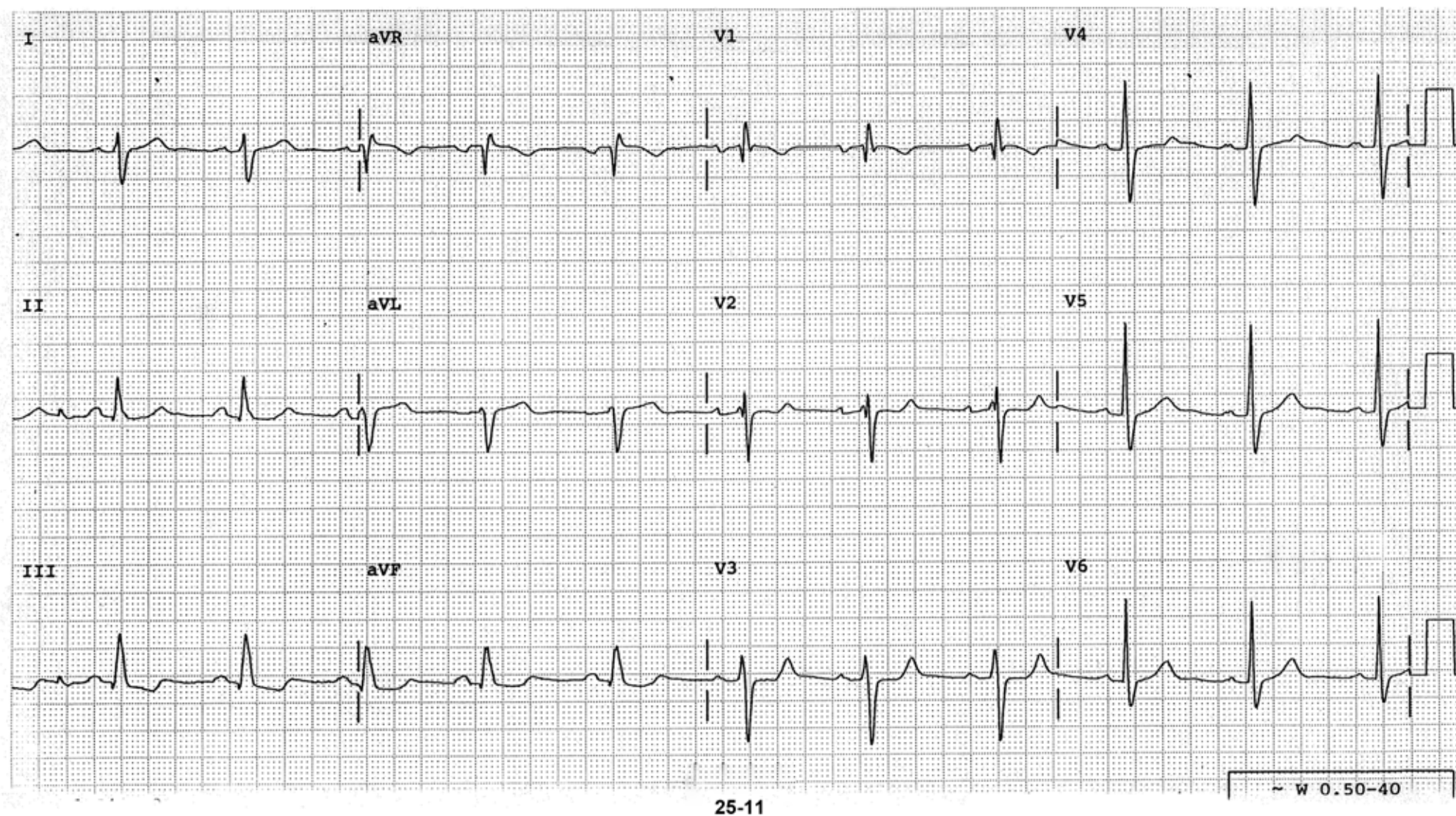
- Dx:
1. Sinus rhythm with 1° AV block
 2. PVCs
 3. LVH with or without old anteroseptal infarct



25-10 Irregular rhythm with an average ventricular rate of about 150/minute. The QRS is narrow. In the inferior leads, an organized P wave can be seen in front of each QRS which occurs irregularly with changing morphology, meeting the criteria of MAT. These P waves are not easily recognizable in other leads, especially in the precordial leads, and this rhythm can easily be mistaken for atrial fibrillation. Diffuse ST-T changes are present. The “scooped-out” configuration of the ST-segment is the effect of digitalis. The R wave is progressing poorly in the precordial leads either due to old anteroseptal infarction, LVH or COPD.

- Dx:*
1. MAT
 2. ST-T changes of digitalis effect
 3. LVH
 4. Poor R wave progression, maybe secondary to LVH, old anteroseptal MI or COPD

25-11 Question: What clinical condition should this tracing make one think of?

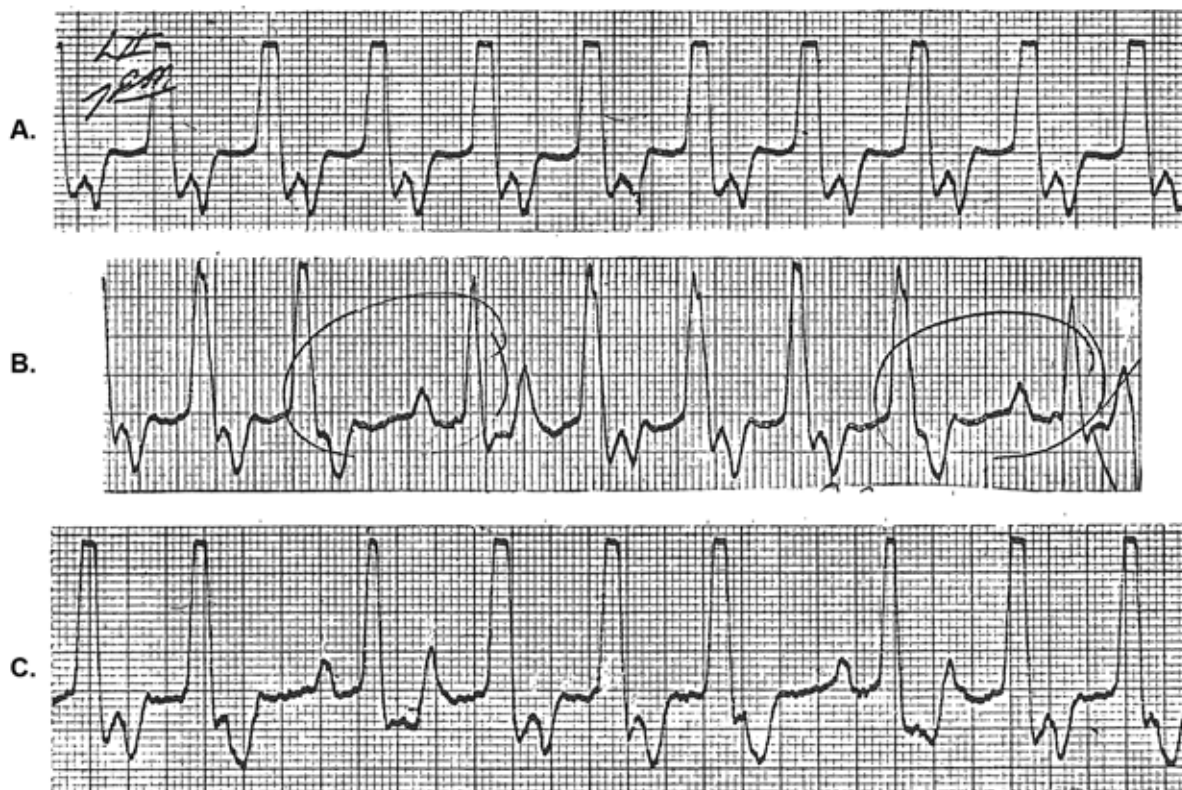


Answer: ASD, secundum

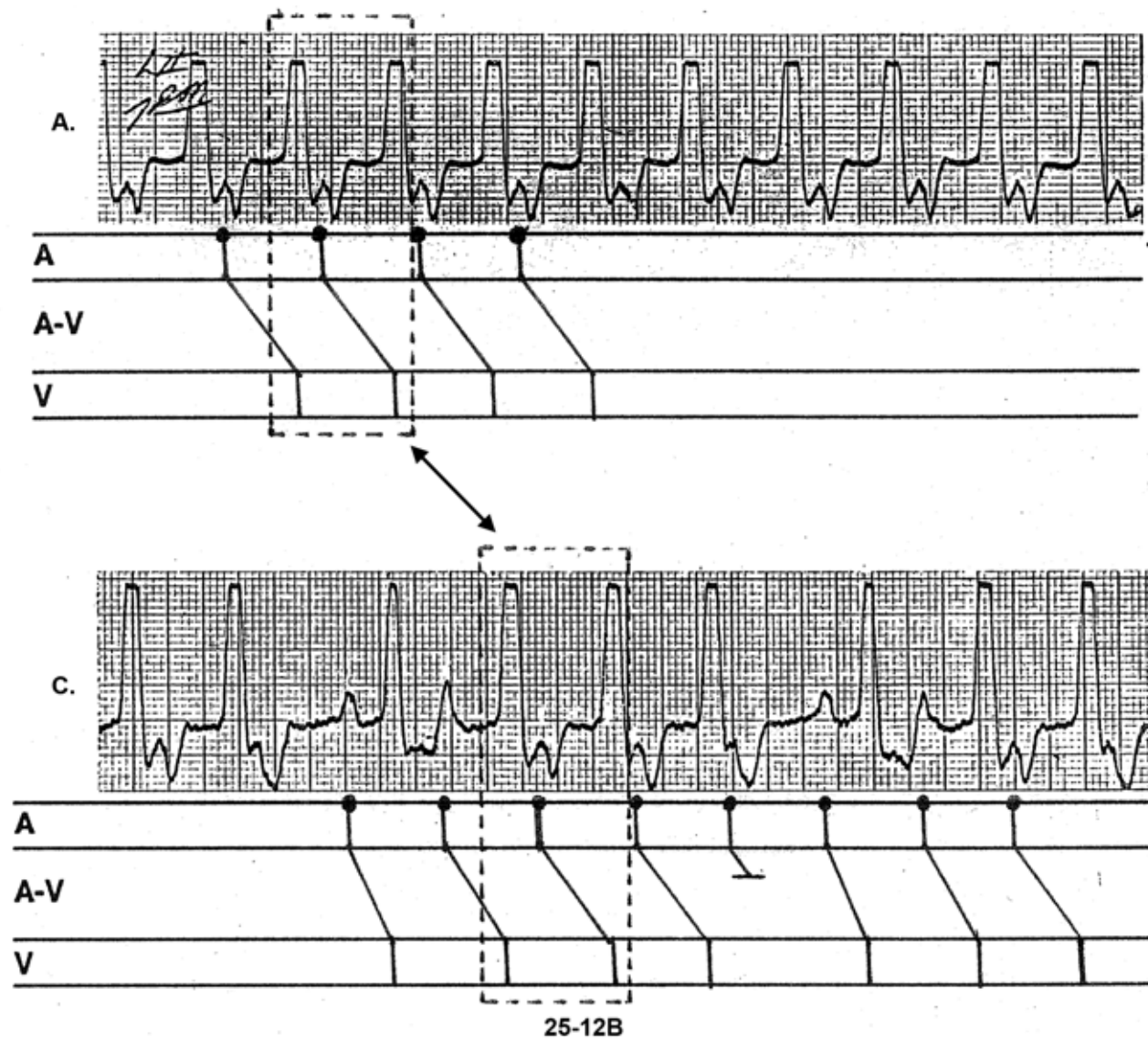
Discussion: The ECG manifestations of secundum ASD are incomplete RBBB and RAD. These findings are felt to be due to RV volume overload. The ECG features of primum ASD are incomplete RBBB and left axis deviation, probably as a result of abnormal development of the left bundle system in the region of the endocardial cushion abnormality. This patient proved to have a secundum ASD.

25-12 Question: Tracings A, B, and C are taken from the same patient. Judging from the tracings B and C, tracing A reveals which of the following? (choose one)

- (A) Sinus tachycardia with 1° AV block and BBB
- (B) Accelerated AV junctional rhythm with a retrograde 1:1 conduction to the atria and BBB
- (C) Acceleration idioventricular rhythm with a retrograde 1:1 VA conduction



25-12A

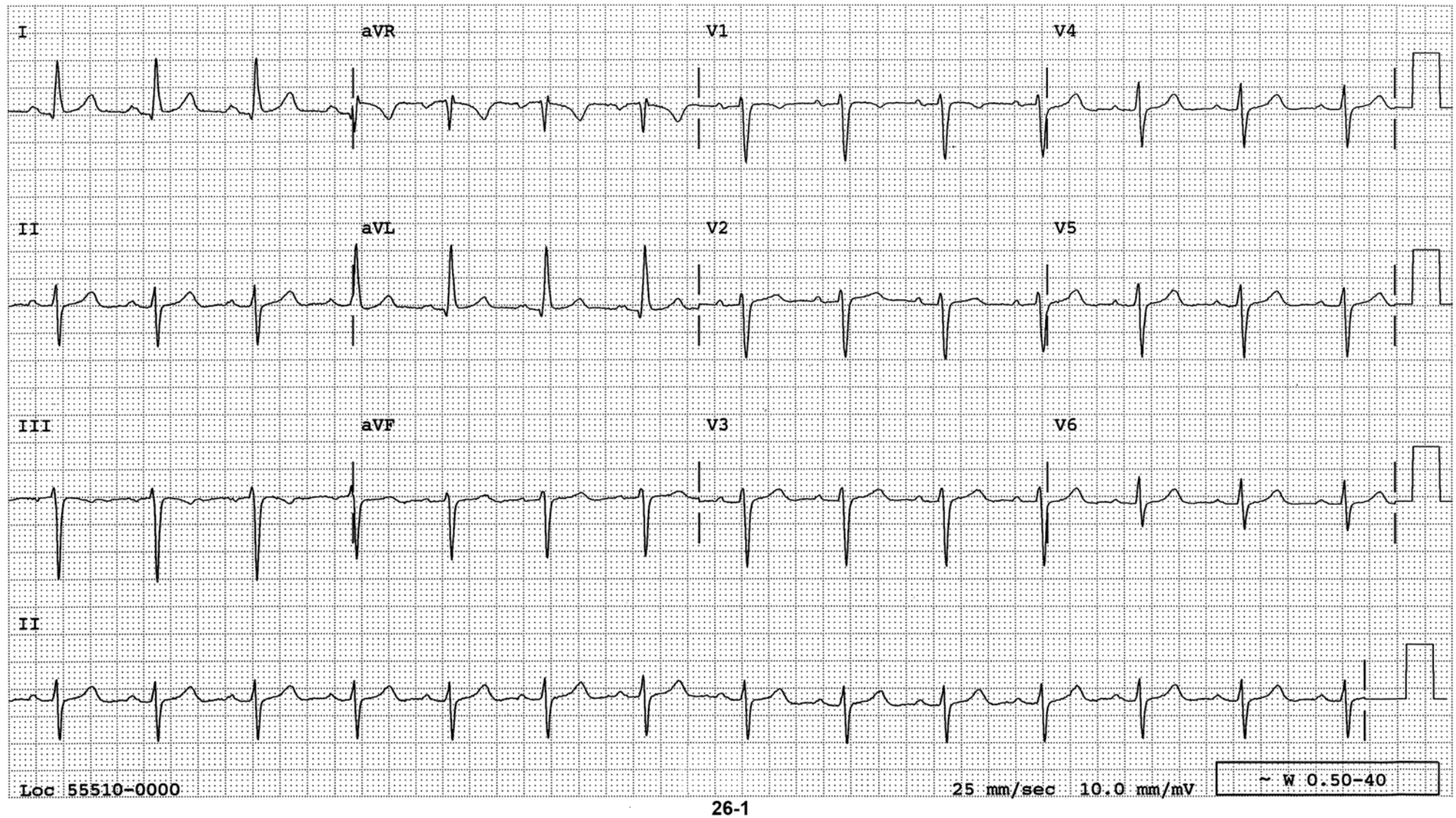


Answer: (A) Sinus tachycardia with 1° AV block and BBB

Discussion: Strip C reveals a sinus rhythm with AV Wenckebach phenomenon and convincingly establishes the P-QRS relationship.

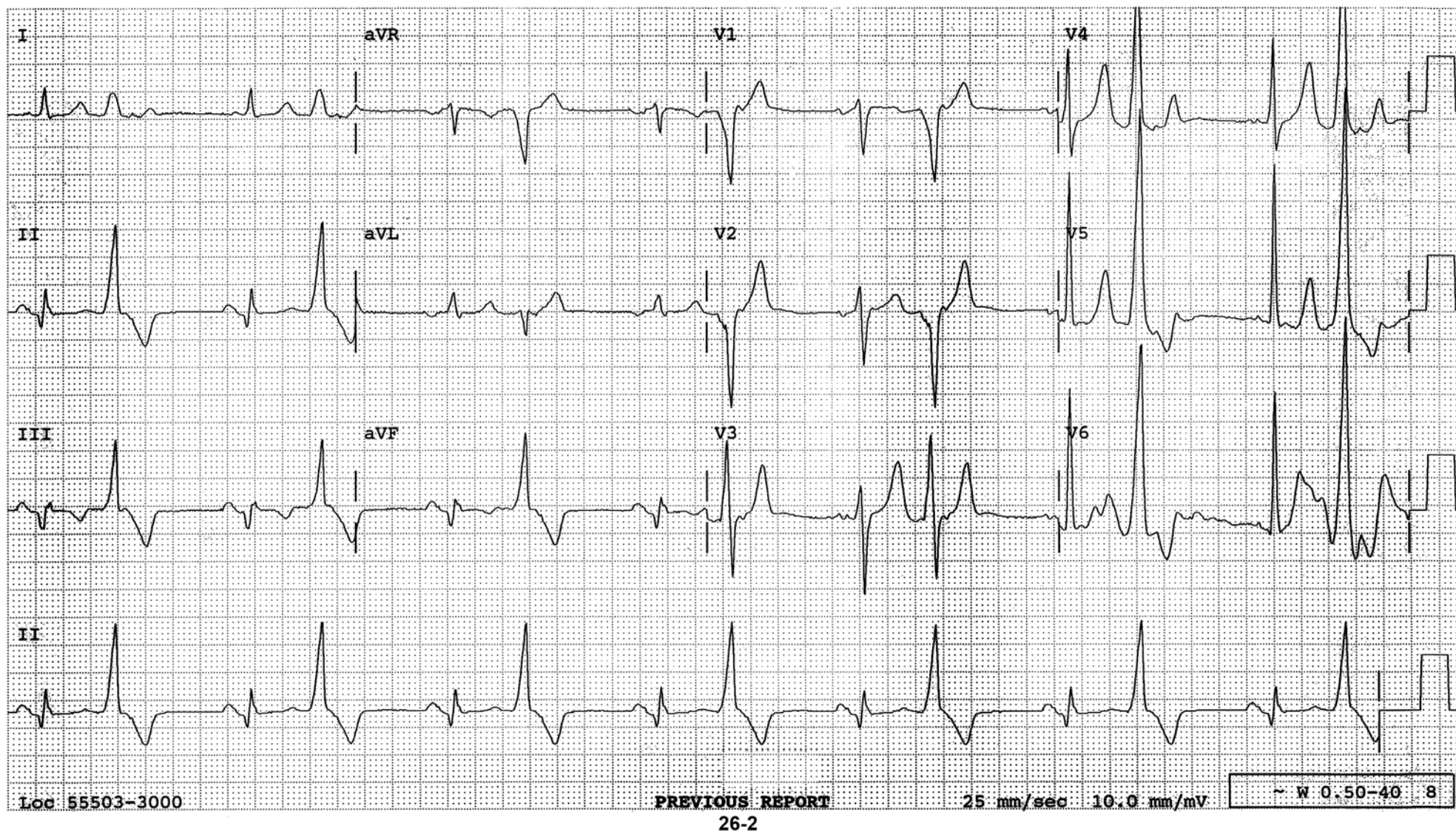
Strip A is made of repetitions of the P-QRS relationship seen within the dotted lines in Strip C. Therefore, it is a sinus rhythm with 1° AV block and BBB.

SECTION 26



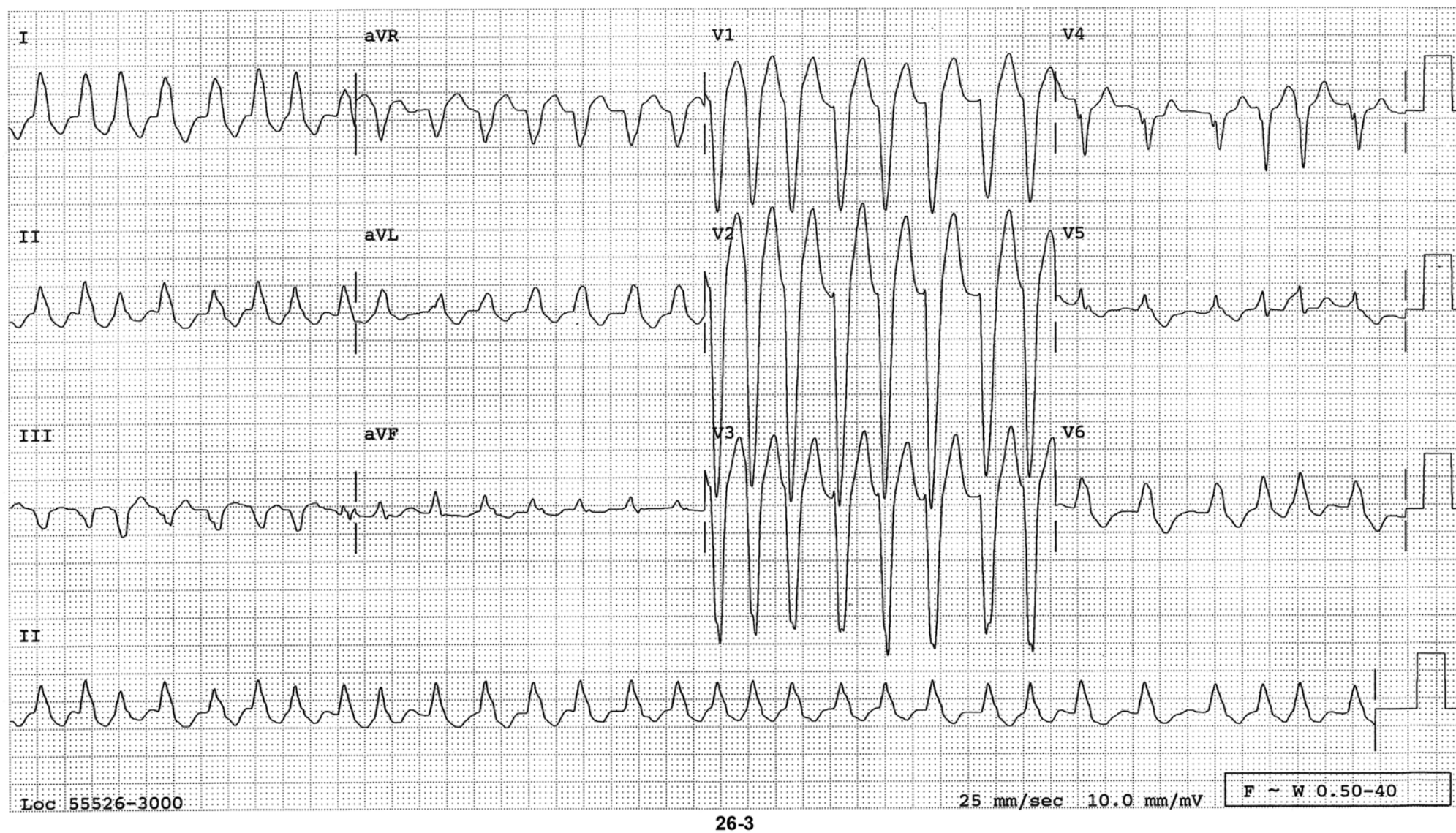
26-1 Normal sinus rhythm at a rate of 82/minute. Left axis deviation is present, indicating left anterior fascicular block. Late transition, as is seen in this tracing, is often associated with LAFB.

- Dx:
1. NSR
 2. LAFB
 3. Late transition



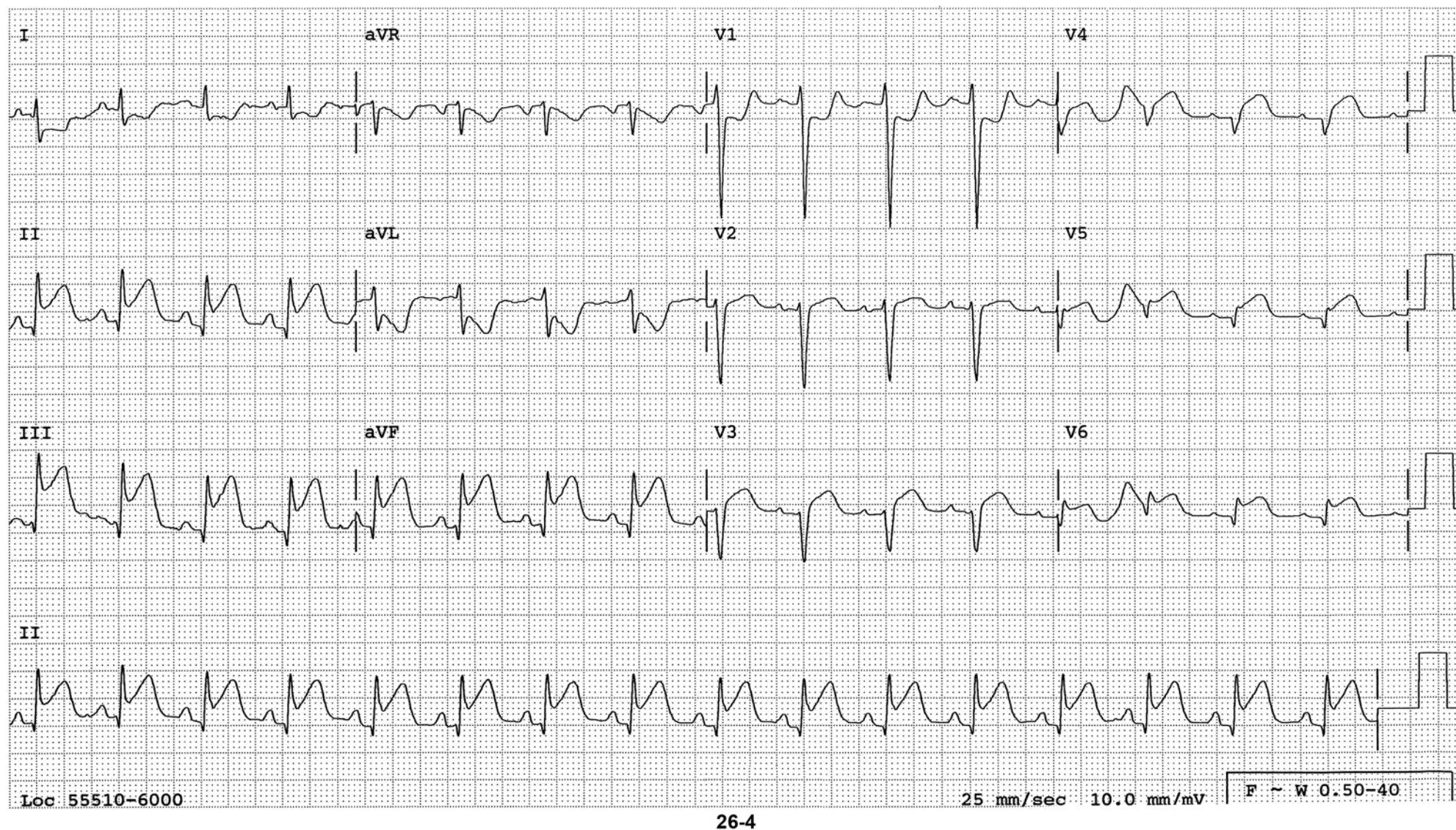
26-2 Normal sinus rhythm with ventricular bigeminy with an average ventricular rate of 90/minute. Q waves with a T wave inversion in the inferior leads indicate recent inferior infarct. Voltage criteria for LVH are present.

- Dx:
1. Sinus rhythm with ventricular bigeminy
 2. LVH by voltage
 3. Recent inferior infarct



26-3 Irregularly irregular rhythm at a rate of 184/minute. QRS is wide and has an LBBB configuration. This tracing can be mistaken for VT, but is an example of atrial fibrillation and LBBB.

- Dx:*
1. Atrial fibrillation with a fast ventricular response
 2. LBBB

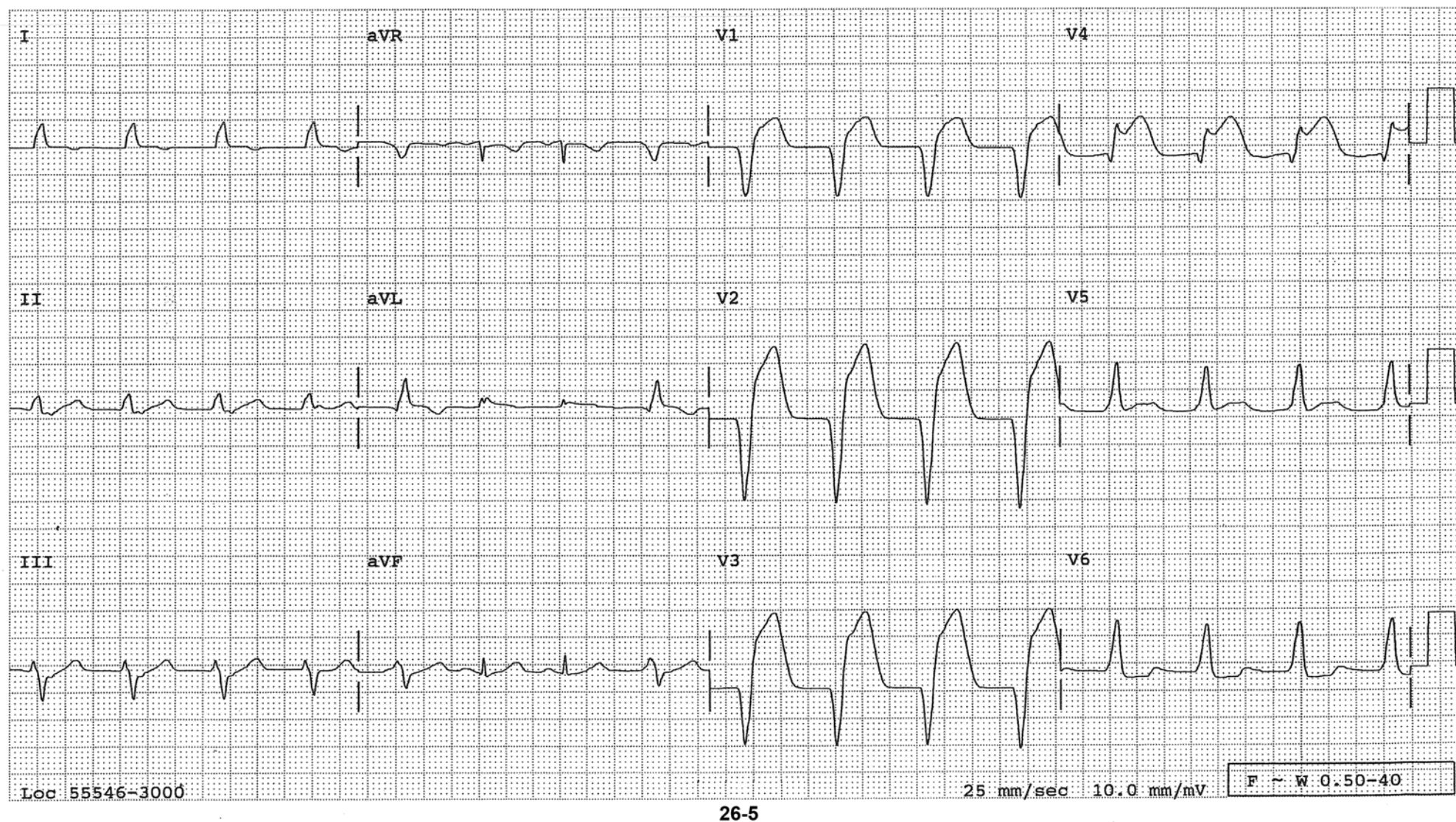


26-4 This tracing shows acute inferoposterior MI and RV infarct

Clues: It's not acute inferior and anterolateral MI. The precordial leads are actually right-sided and reveal RV infarct (ST elevation in RV_{3-6}). Ordinarily, there are two clues for the right-sided precordial leads:

1. Biphasic or entirely negative P waves in V_4 - V_6
2. QRS vector in V_6 is opposite from that of leads I and aVL

In this patient, the P wave morphology does not help. In fact, it could be misleading because it is entirely positive in V_4 - V_6 , which is unusual. However, the QRS vector in V_6 is opposite from that of leads I and aVL, indicating that these are not regular left-sided precordial leads. Again, ST depression in lead I already indicated RV involvement. A significant horizontal ST depression in V_1 , which is regular V_2 , indicates posterior wall involvement as well.

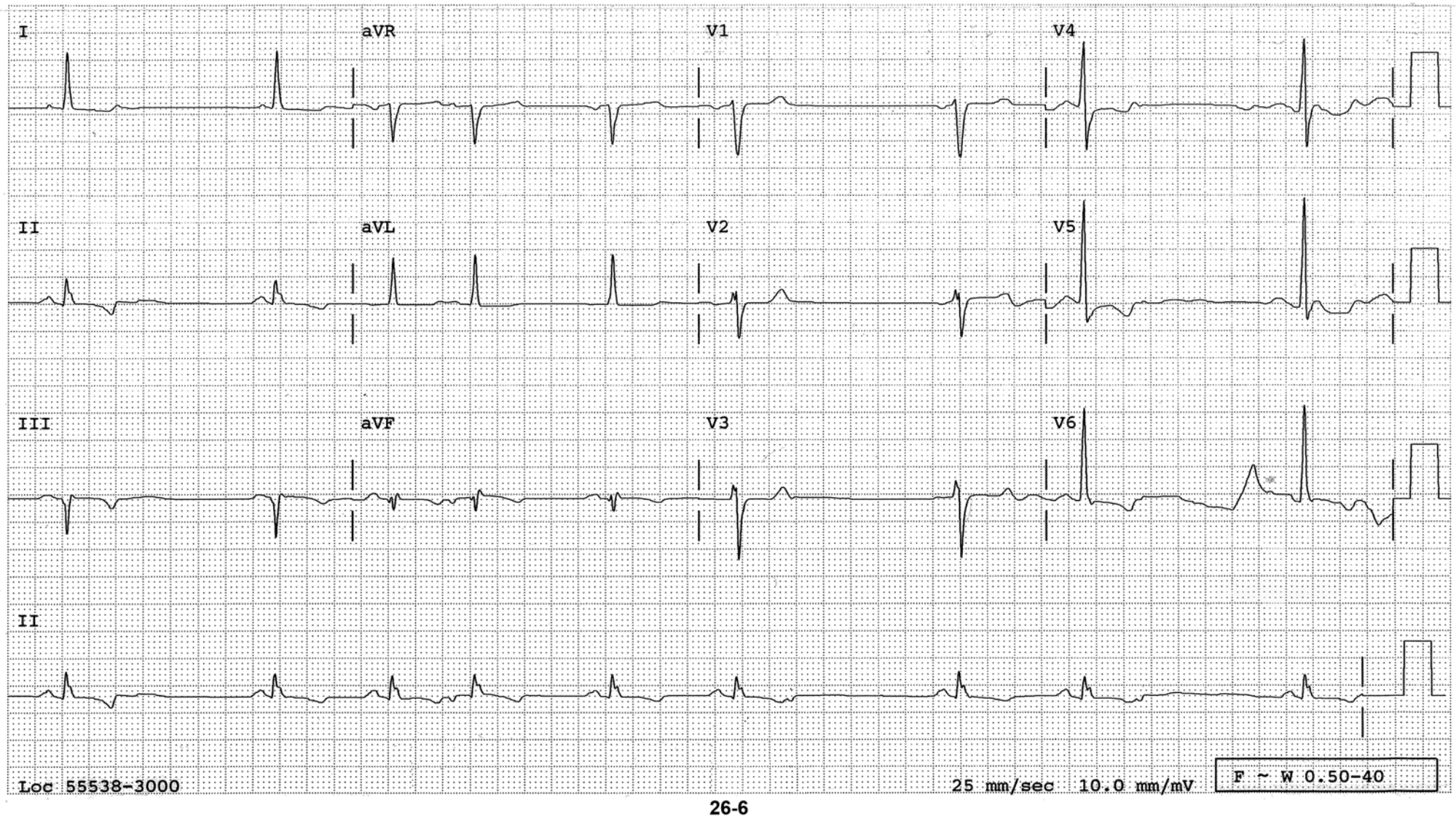


26-5 Regular wide QRS rhythm at a rate of 85/minute. No P waves are seen and the rhythm is accelerated idioventricular rhythm (two beats in aVR, aVL and aVF are normal sinus beats). The qR pattern with ST elevation in V_4 is diagnostic of STEMI, which is what this patient has. Thus, even QRSs originating from the ventricle such as PVCs, QRSs in VT, accelerated idioventricular rhythm and ventricularly paced rhythm can at times reveal STEMI very effectively.

Dx: Accelerated idioventricular rhythm revealing STEMI of anterior wall very effectively

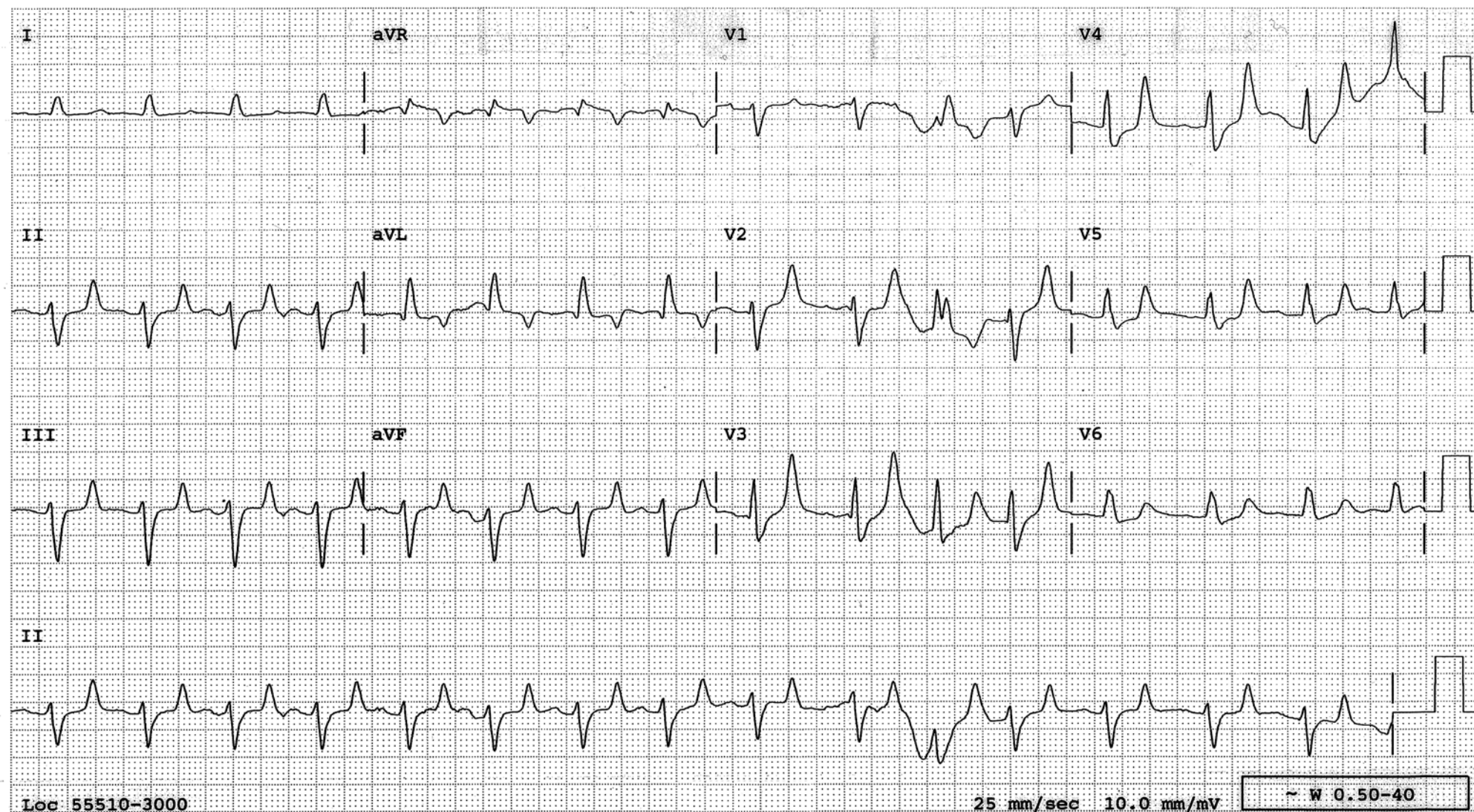
26-6 Question: The pauses in this tracing reflect (choose one from below):

- (A) Nonconducted atrial premature depolarization
- (B) Sinus node dysfunction
- (C) Sinus arrhythmia
- (D) Type II 2° AV block



Answer: (A) Nonconducted atrial premature depolarization

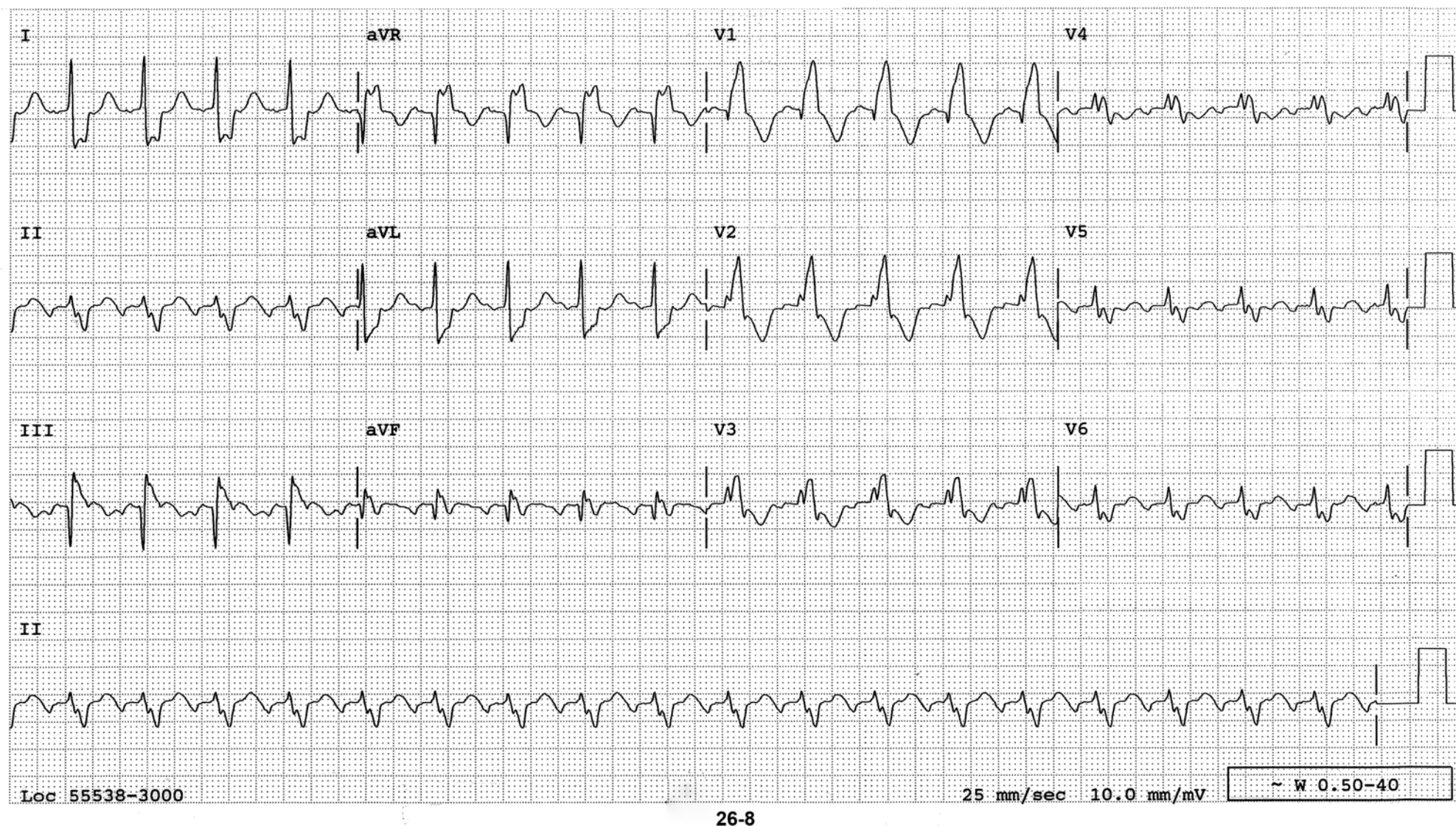
Discussion: Many pauses are present. There is a blip near the end of the T wave of the second and fourth QRSs from the end, which is good for an atrial activity, indicating atrial premature depolarizations that are not conducted to the ventricles. In fact, the fourth QRS is clearly a conducted atrial premature beat, supporting that interpretation. Whenever one encounters unexpected pauses, hidden P waves that are not conducted should be looked for. The Q wave in lead III is wide and deep enough, indicating inferior infarct. Nonspecific ST changes are also present.



26-7

26-7 Sinus rhythm at a rate of 90/minute. P waves are present in front of each QRS in the rhythm strip of lead II, but they are flat. The QRS is wide and measures about 140 milliseconds. The T waves are tall, narrow and pointed in the mid precordial leads. These findings are highly suggestive of hyperkalemia.

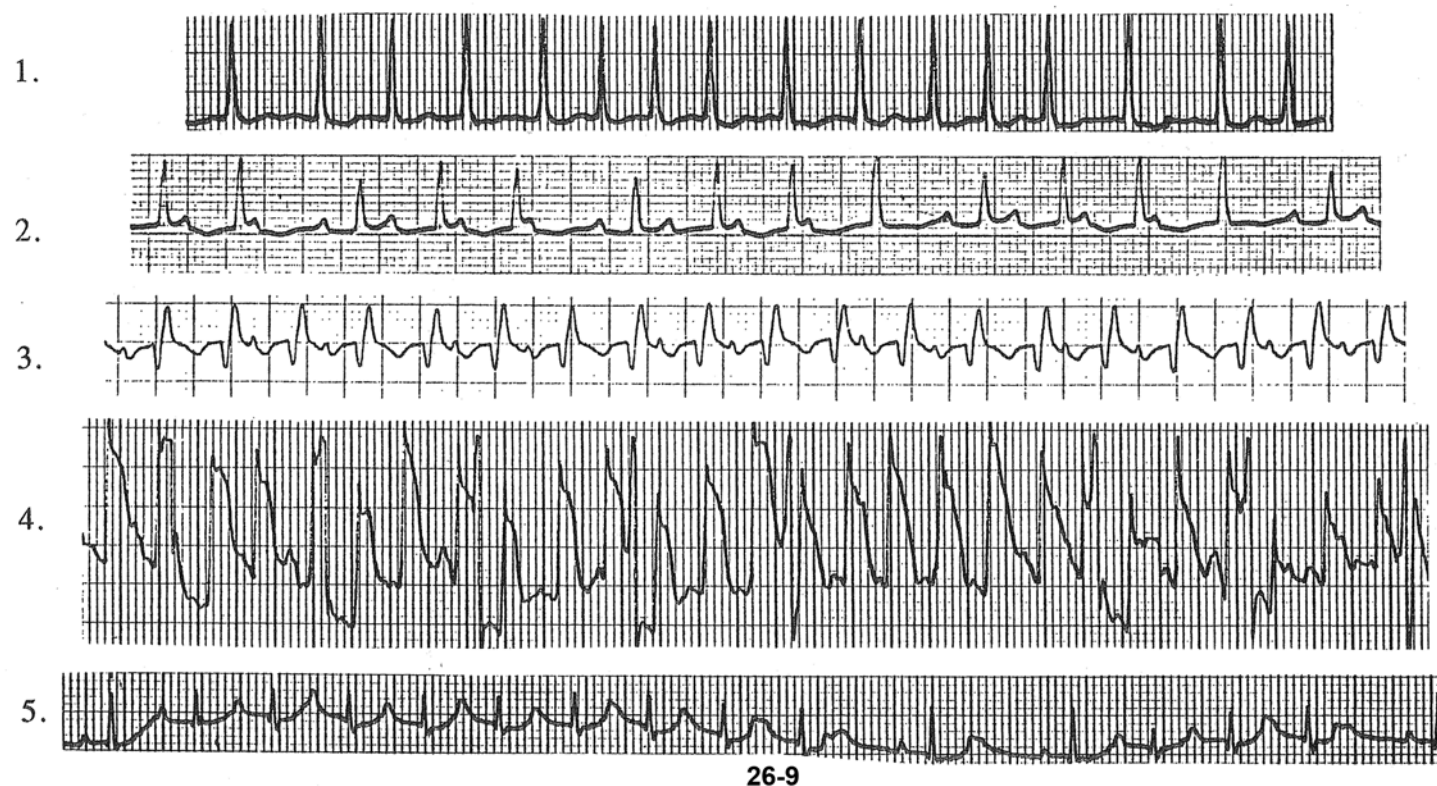
- Dx: 1. Sinus rhythm
2. Hyperkalemia



26-8 The “sawtooth” pattern of atrial flutter is obvious in the rhythm strip of lead II. The flutter rate is somewhat slow at 240/minute. Antiarrhythmics are well-known to slow the flutter rate down to 200/minute very easily. Complete RBBB is also present. Q waves in lead III and aVF indicate inferior infarct as well.

- Dx:*
1. Atrial flutter with 2:1 AV conduction
 2. RBBB
 3. Inferior MI, probably old

26-9 Question: What do these rhythm strips show?



26-9

Answer: Tracing 1 is an irregularly irregular rhythm with no organized P waves and is typical of atrial fibrillation

Tracing 2 reveals P waves occurring regularly at a rate of 150/minute. Group beatings of the QRSs and the P-R interval progressively lengthening until, eventually, a P wave is blocked, indicate atrial tachycardia with AV Wenckebach phenomenon.

Tracing 3 reveals wide QRS tachycardia at a rate of 160/minute. Two QRSs are followed by a blip, consistent with a P wave, while the third QRS is not followed by a blip. Additionally, the QRS-to-blip interval is short with the first beat, and longer with the second QRS, reflecting VT with 3:2 retrograde VA Wenckebach phenomenon.

Tracing 4 at first glance may appear to be a run of VT or ventricular flutter. However, regularly occurring slower narrow deflections are recognizable, and this is an example of artifact mimicking a run of VT during sinus rhythm.

Tracing 5 reveals regularly occurring atrial activities at a rate of 150/minute. The P-R interval progressively lengthens until one is finally blocked. After one stretch of 2:1 AV block, a Wenckebach cycle begins again. Atrial tachycardia with variable AV conduction should raise the possibility of digitalis toxicity.

26-10 *Question:* Match the tracings from patient A and patient B with the clinical descriptions (a) and (b) below:

(a) Blood pressure which decreases by 20 mm Hg with inspiration

(b) Taking digitalis and is nauseated

Patient A



Patient B



26-10

Answer: Patient A: (b); Patient B: (a)

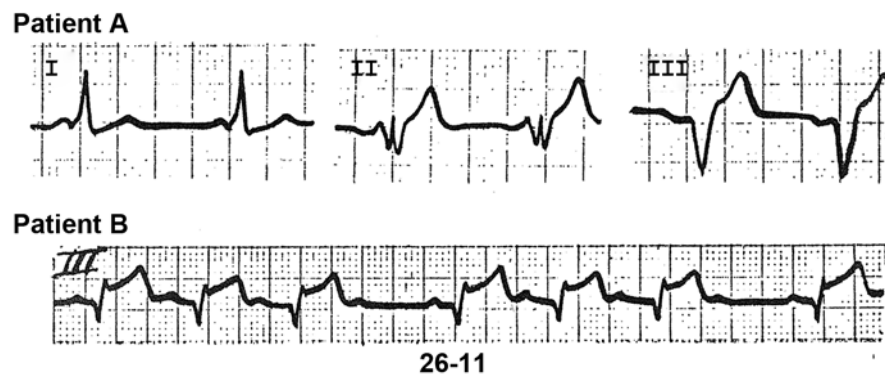
Discussion: The tracings from patient A and patient B have a similarity in that every other QRS vector is opposite from the other. In the tracing of patient A, the QRSs are wide with no P waves and is an example of bidirectional tachycardia which is highly suggestive of digitalis toxicity. It is either junctional tachycardia which is conducted through alternating bundle branches or VT utilizing alternating conduction pathways. This can be mistaken for ventricular bigeminy, but the fact that the R-R interval is same rather than long and short proves that it is not ventricular bigeminy.

The tracing in patient B is a sinus tachycardia with narrow QRS and is an example of electrical alternans (EA). Electrical alternans during sinus rhythm is highly specific for cardiac tamponade. Electrical alternans in tamponade is well-documented to be due to a pendular motion of the heart with each cardiac cycle so that with one cycle, the heart is much closer to the chest wall and with the other cycle, the heart is away from the chest wall. It is well known that some cases of SVT, atrial flutter and VT may be associated with electrical alternans. In that case, cardiac tamponade is not present.

26-11 *Question:* Match the tracings from patient A and patient B with the clinical descriptions (a) and (b) below.

(a) Episodes of palpitations

(b) Crushing substernal chest pain

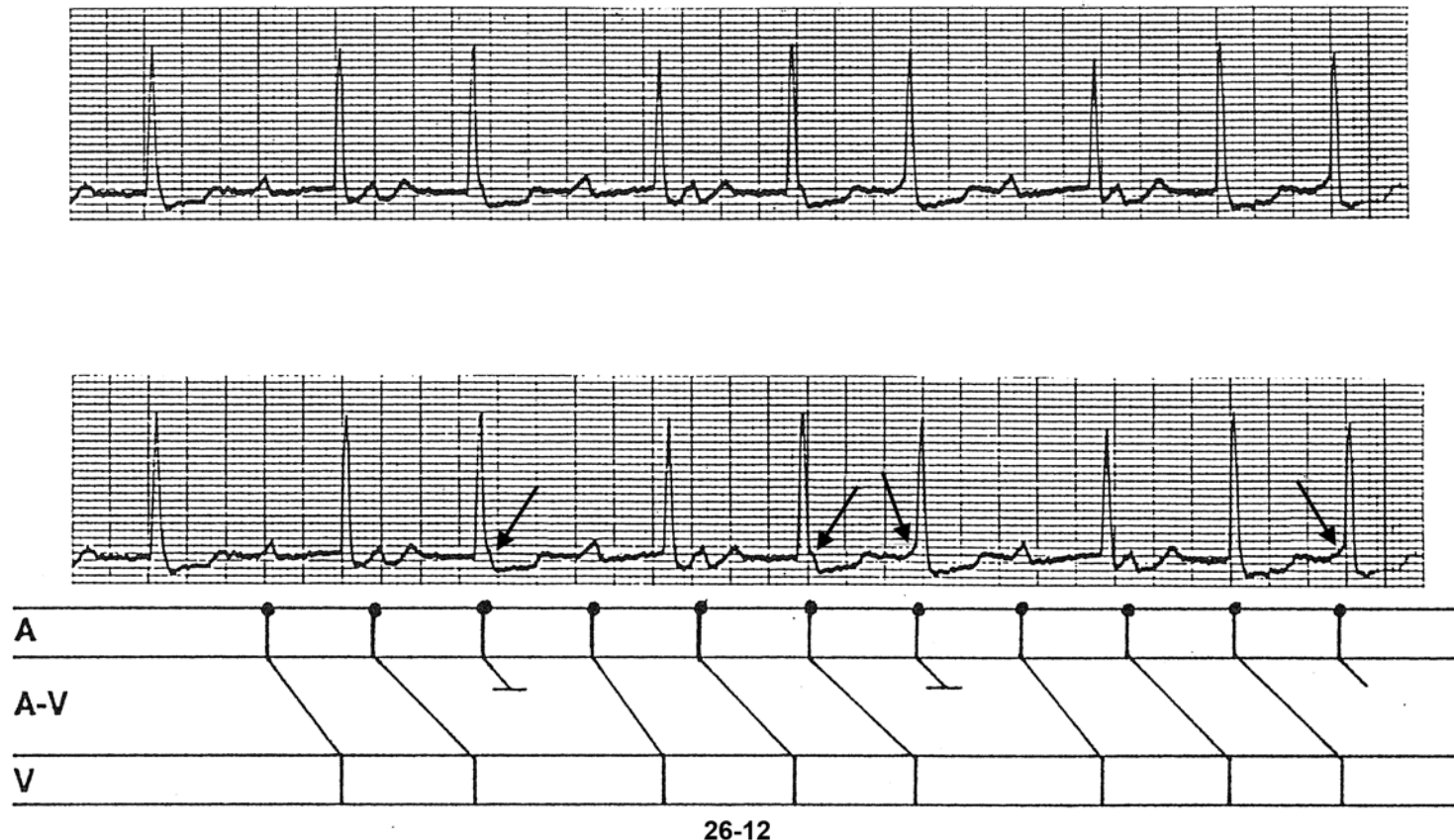


Answer: Patient A: (a); Patient B: (b)

Discussion: Patient A is the one with palpitations, and the tracing reveals WPW syndrome. The delta wave is superiorly oriented, registering as a negative wave in the inferior leads, mimicking an inferior MI. Patient B is the one with crushing chest pain who has Q waves with ST elevation in the inferior leads. The tracing also reveals Mobitz Type I 2° AV block (Wenckebach phenomenon), which inferior MI is known to do.

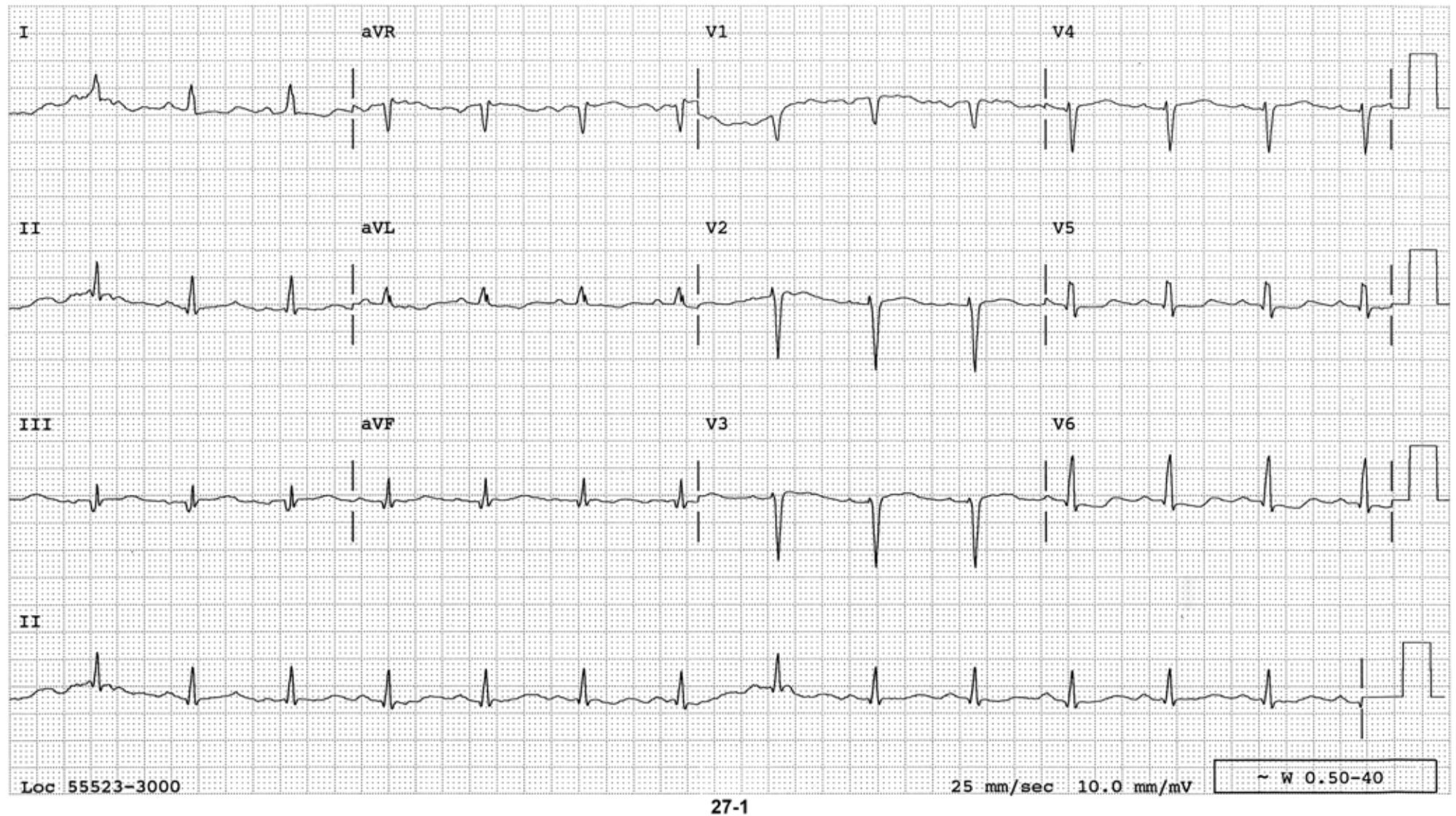
26-12 Question: This tracing reveals (choose one from below):

- (a) NSR with 1° AV block and occasional sinus node dysfunction (sinus arrest or SA block)
- (b) NSR with AV Wenckebach phenomenon
- (c) NSR with 3° AV block
- (d) NSR with occasional sinus node dysfunction and 3° AV block



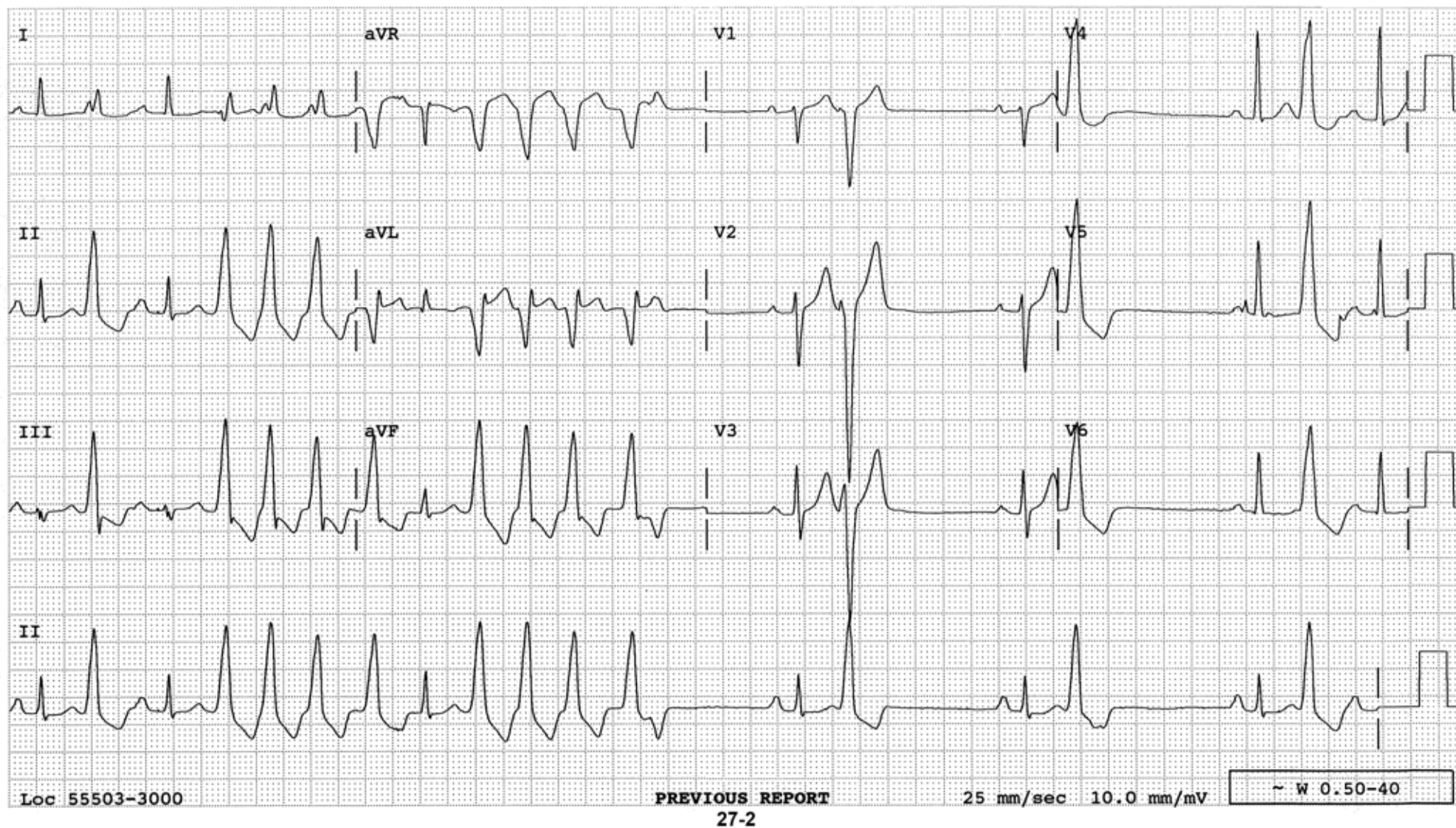
Answer: (b) Normal sinus rhythm with a Mobitz Type I 2° AV block (AV Wenckebach phenomenon) as diagrammed
QRSs do not occur regularly, ruling out 3° AV block right away. P waves occur regularly ruling out (a) above.

SECTION 27



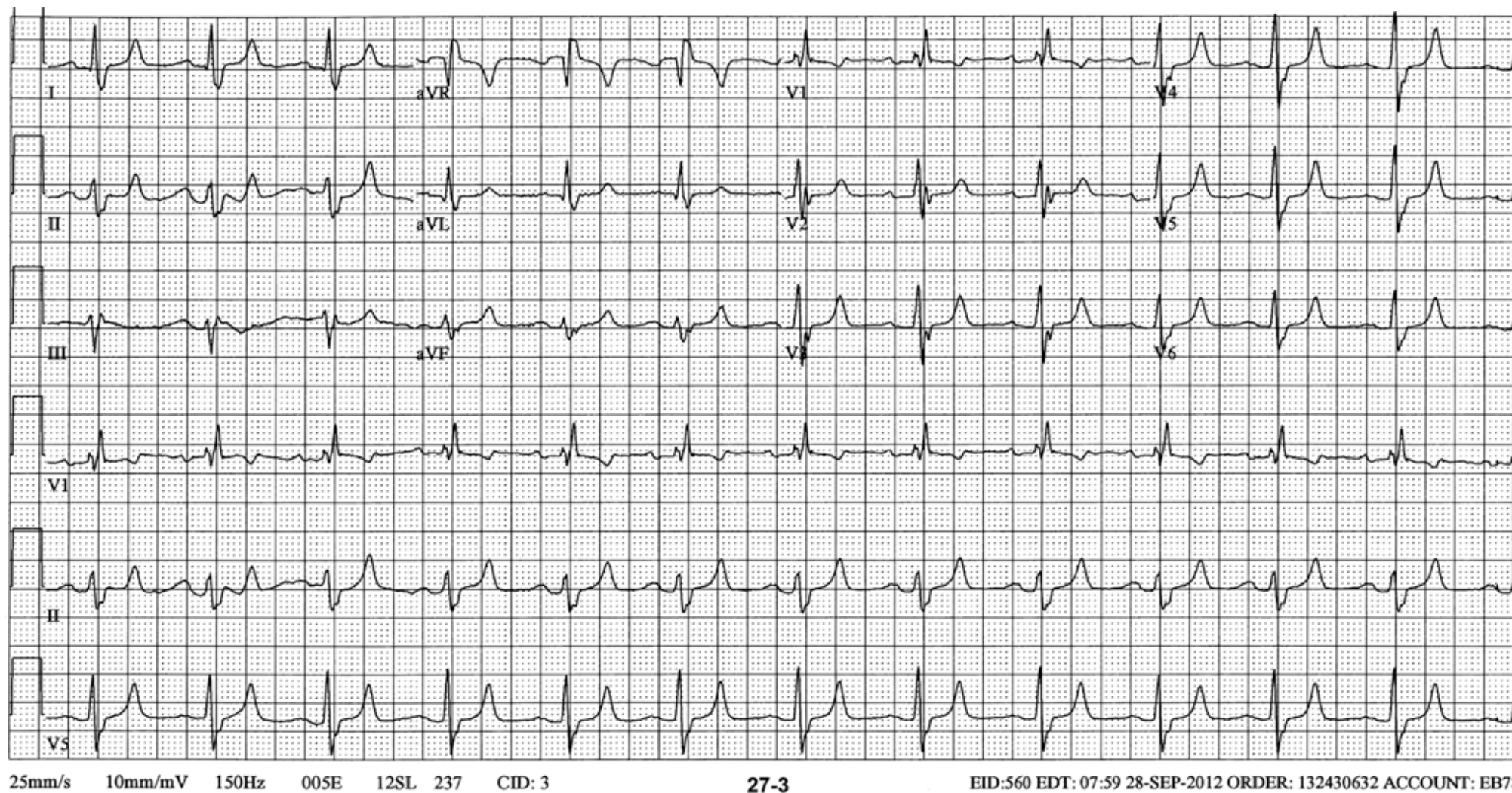
27-1 Normal sinus rhythm at a rate of 84/minute. The Q wave in lead III may not appear impressive, but it is certainly wide enough and deep enough to indicate old inferior infarct. There is poor R wave progression in the right precordial leads which may or may not be due to old AMI. ST-T changes are present.

- Dx:*
1. NSR
 2. Old inferior infarct
 3. Poor R wave progression
 4. Nonspecific ST-T changes



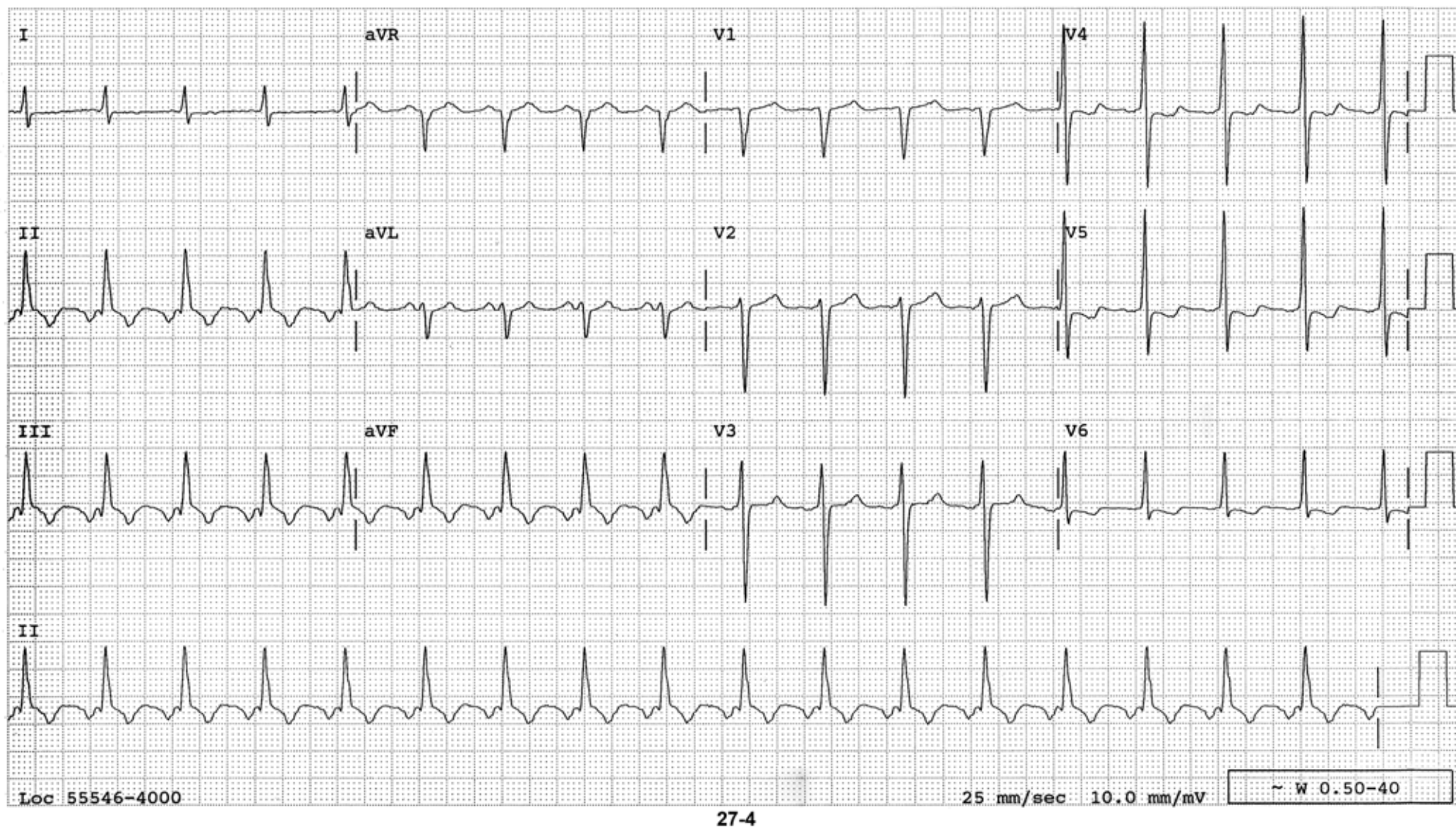
27-2 Normal sinus rhythm at a rate of 63/minute. Frequent PVCs and short runs of VT are present. QR pattern in aVL, even during VT, suggests MI.

- Dx:
1. NSR
 2. Frequent PVCs with short runs of VT
 3. Consider high lateral MI



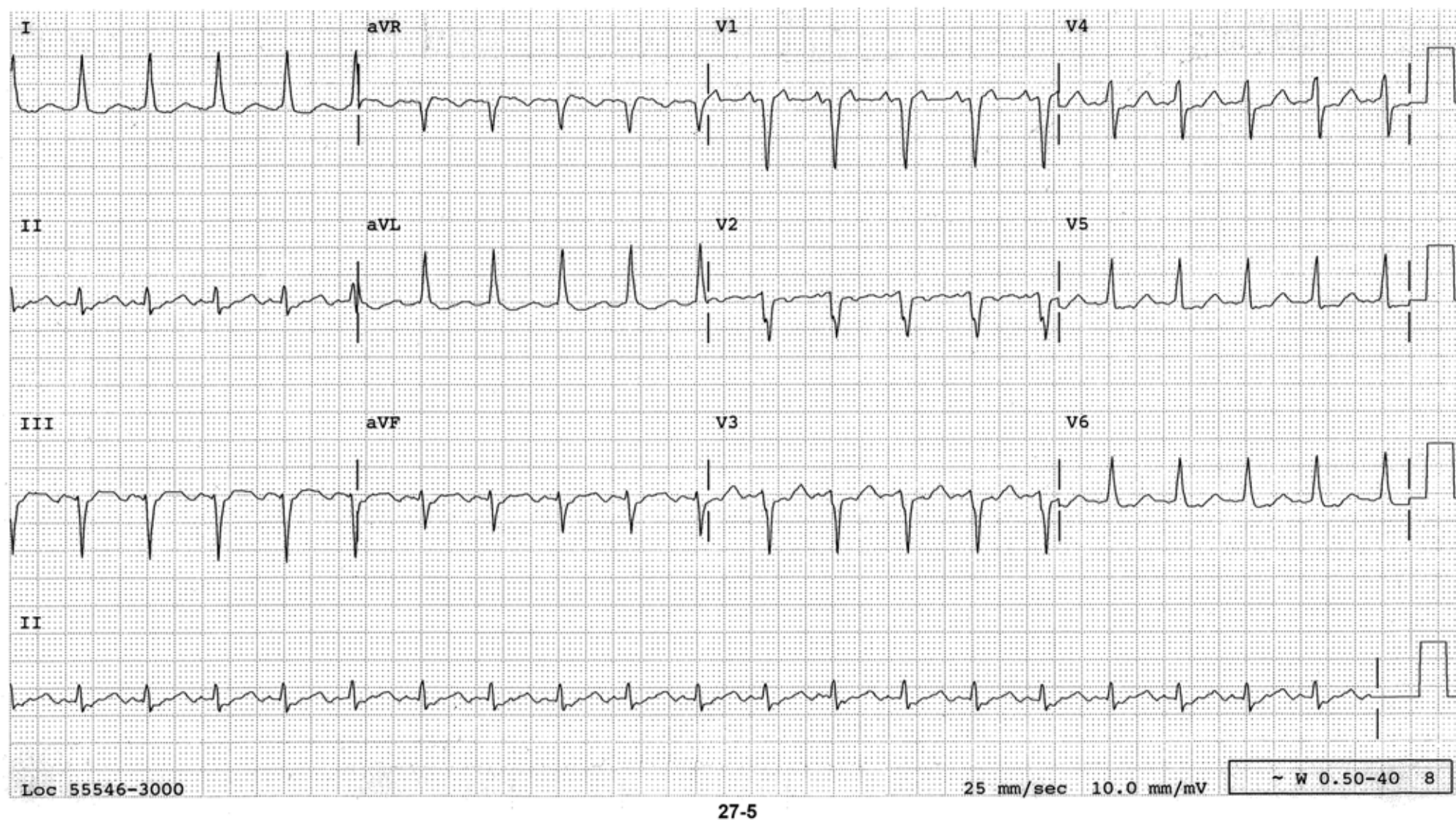
27-3 Normal sinus rhythm at 75/min. RBBB is present. T waves are not tall but narrow, tented and pointed, reflecting hyperkalemia. The serum K was 6.8 mEq/L. Thus, the T waves do not have to be tall to consider hyperkalemia.

- Dx:*
1. NSR
 2. RBBB
 3. Hyperkalemia



27-4 Atrial flutter waves are easily recognizable in the rhythm strip of lead II. The flutter rate is slow at about 200/minute, suggesting that the patient is taking Type Ia or Ic antiarrhythmic drugs, or the atrial size is huge. Nonspecific ST changes are present.

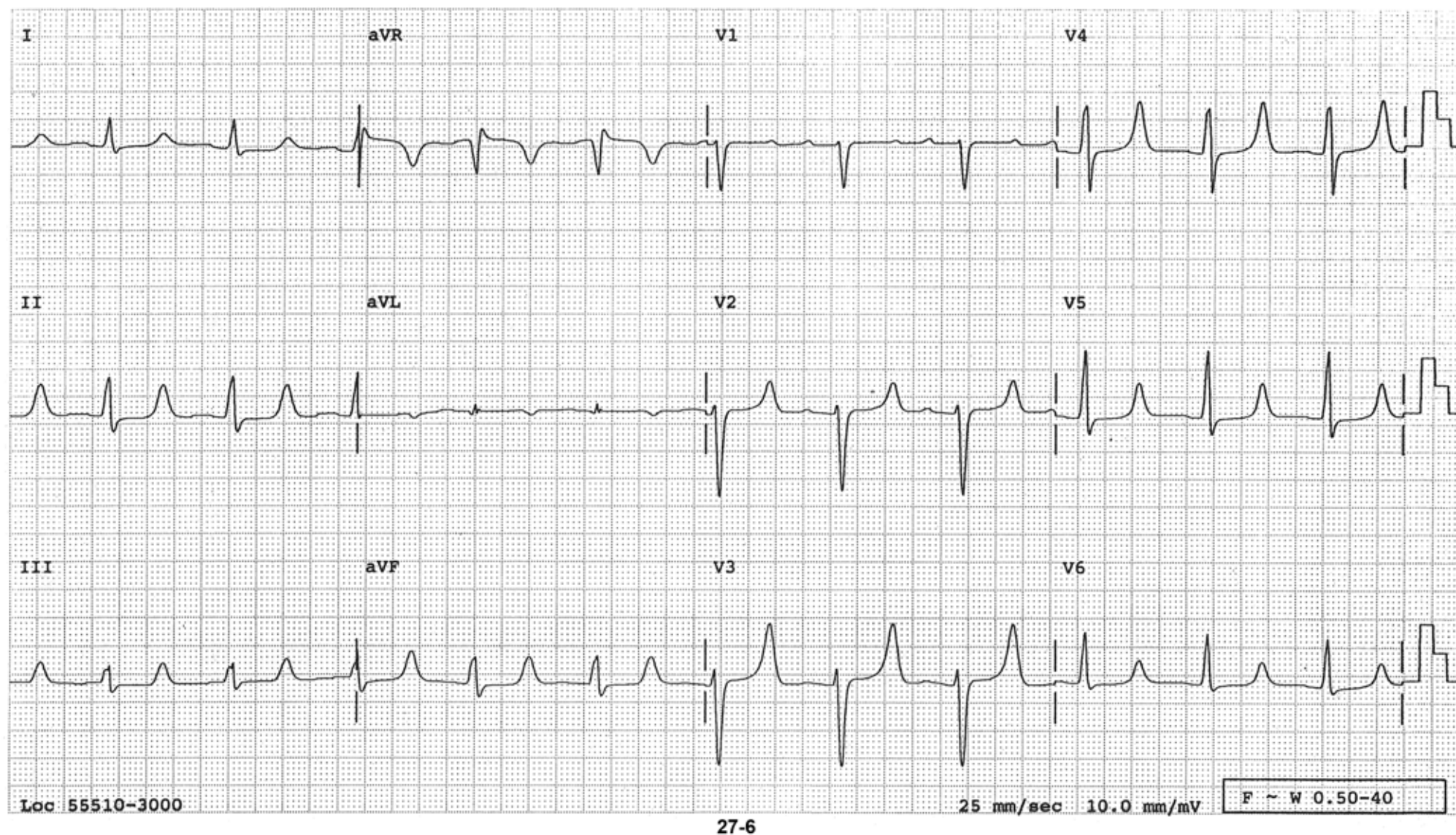
- Dx:
1. Atrial flutter with 2:1 AV conduction with a slower flutter rate of 200/minute
 2. Nonspecific ST-T changes



- 27-5 Regular narrow QRS tachycardia at a rate of 125/minute. V_1 reveals two atrial activities for each QRS. (The blip immediately following the QRS peaks too early for it to be a T wave.) These atrial activities occur regularly at a rate twice that of QRS, making this tracing either atrial tachycardia with 2:1 AV conduction or atrial flutter with 2:1 AV conduction. Armed with this information, one can make out the sawtooth pattern of atrial flutter in inferior leads as well as in lead I and aVL. If this were atrial tachycardia with 2:1 AV conduction, one would have expected to see discrete P waves with an isoelectric baseline in between even in the inferior leads as in V_1 . Instead, the baseline continuously slopes up, then slopes down, and is characteristic of flutter waves. The atrial rate of 250/minute also favors atrial flutter which is too fast for atrial tachycardia.

Dx: Atrial flutter with 2:1 AV conduction

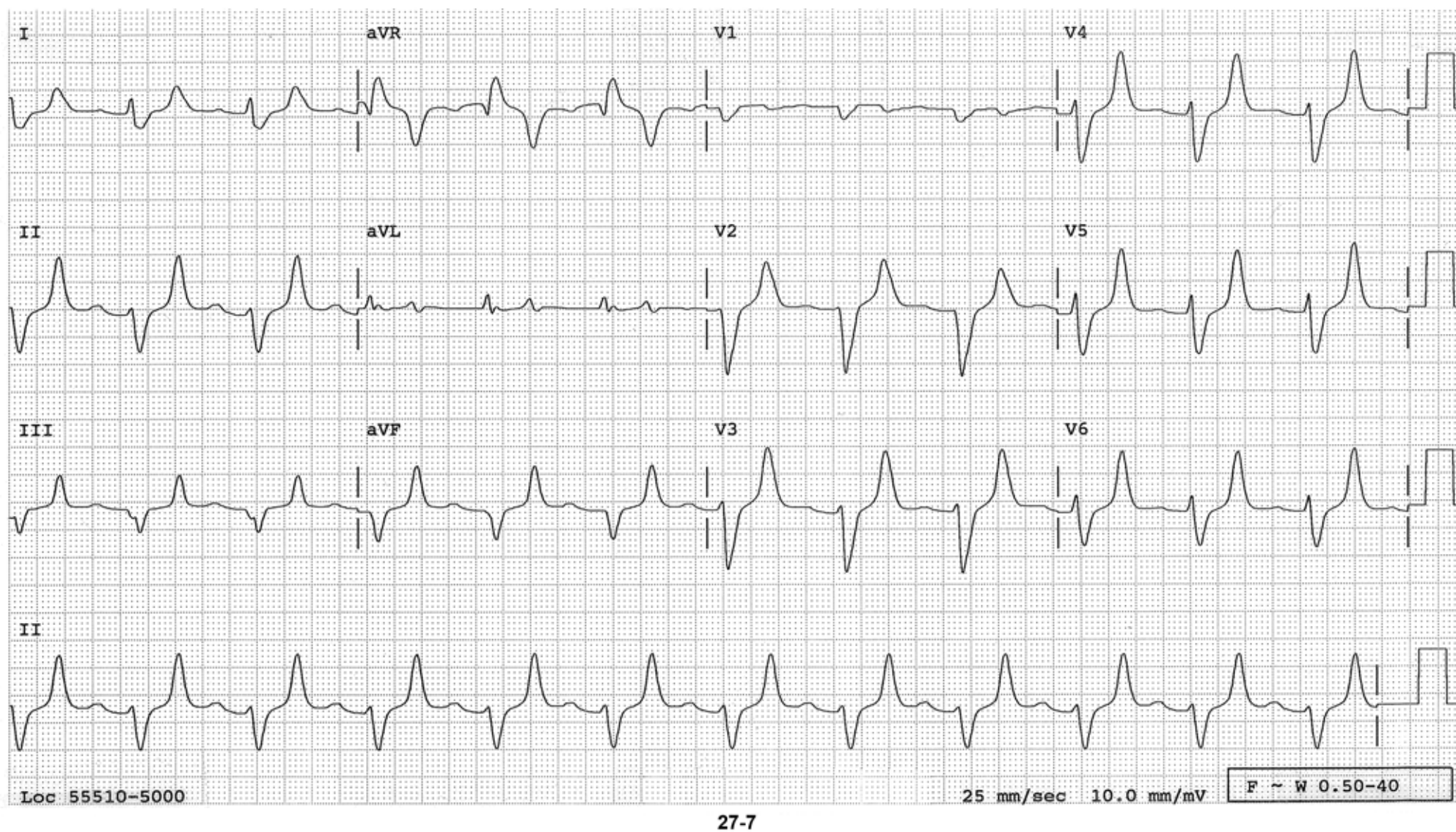
27-6 Question: What electrolyte abnormalities should this tracing make one think of?



Answer: Hyperkalemia and hypocalcemia

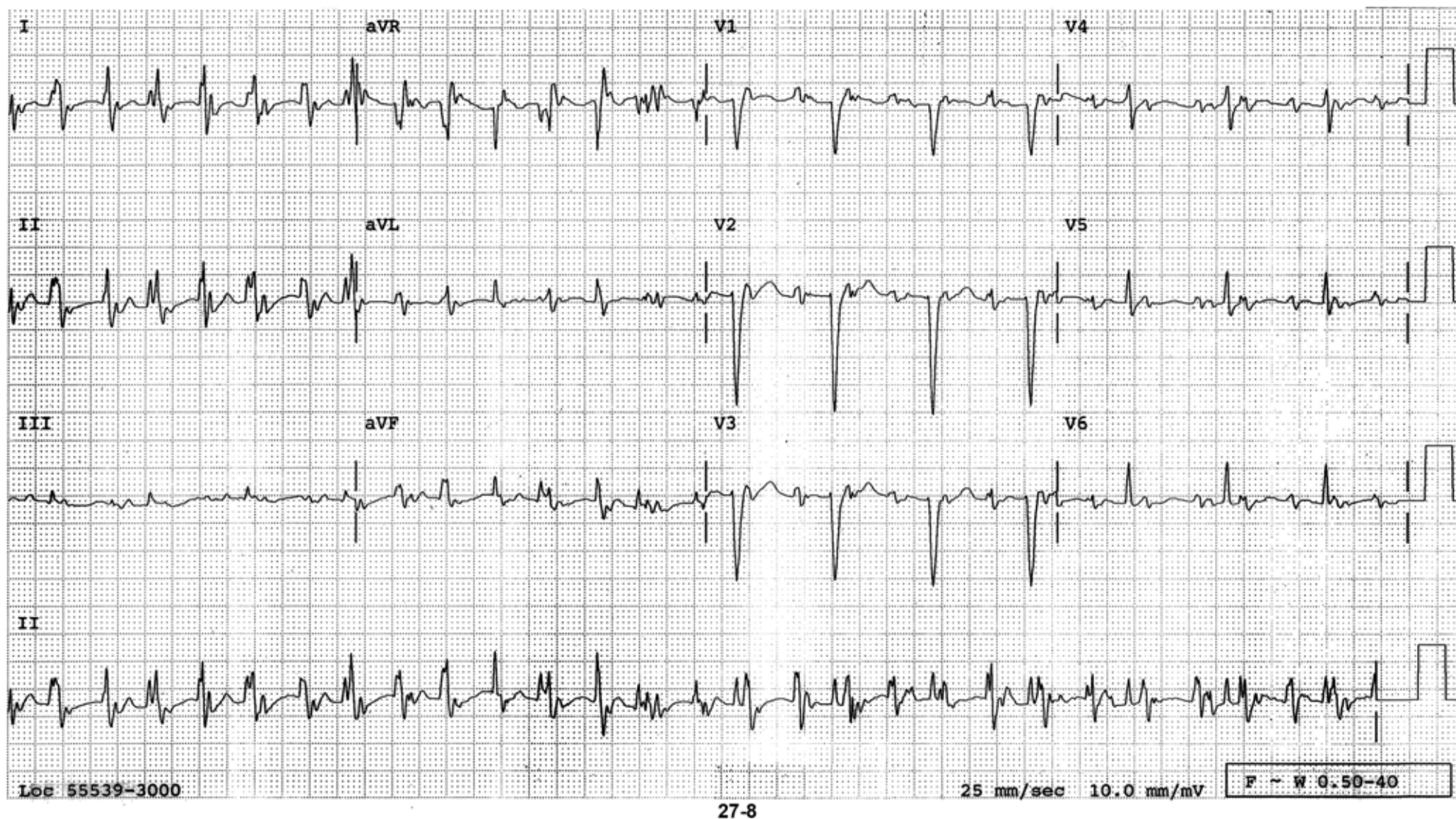
Discussion: Hyperkalemia causes tall, pointed and narrow T waves. Hypocalcemia lengthens the ST-segment with a delayed onset of the T wave and, consequently, a long Q-T interval. The combination of these two electrolyte abnormalities is often seen in patients with renal failure which is what this patient has.

27-7 Question: What clinical condition should this ECG tracing make you think of?



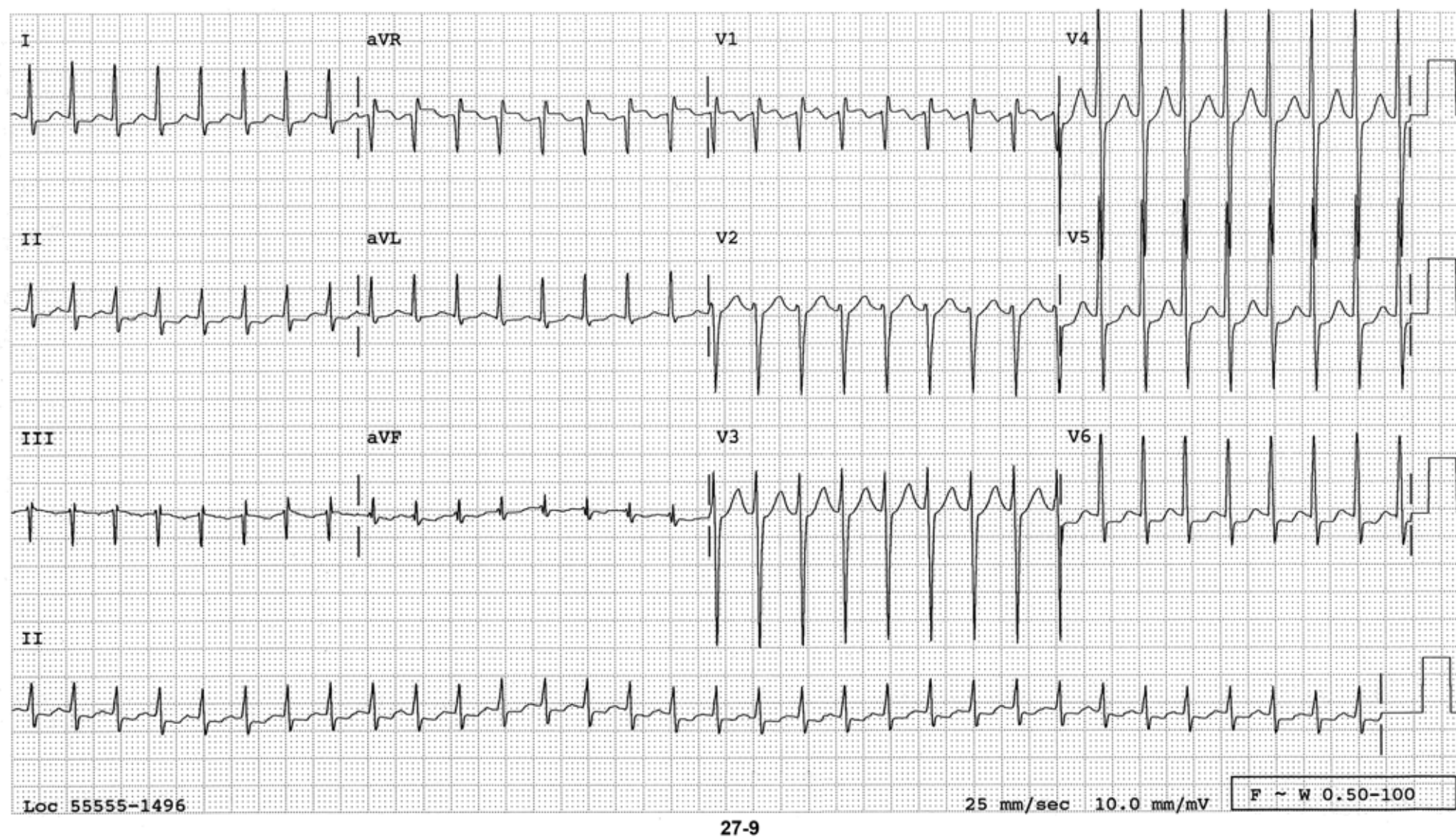
Answer: Hyperkalemia

Discussion: Normal sinus rhythm at a rate of 70/minute. The P waves tend to be flat in many leads. The P-R interval is prolonged. The QRS duration is increased and the T waves are tall, tented, narrow and pointed. These findings strongly suggest hyperkalemia.



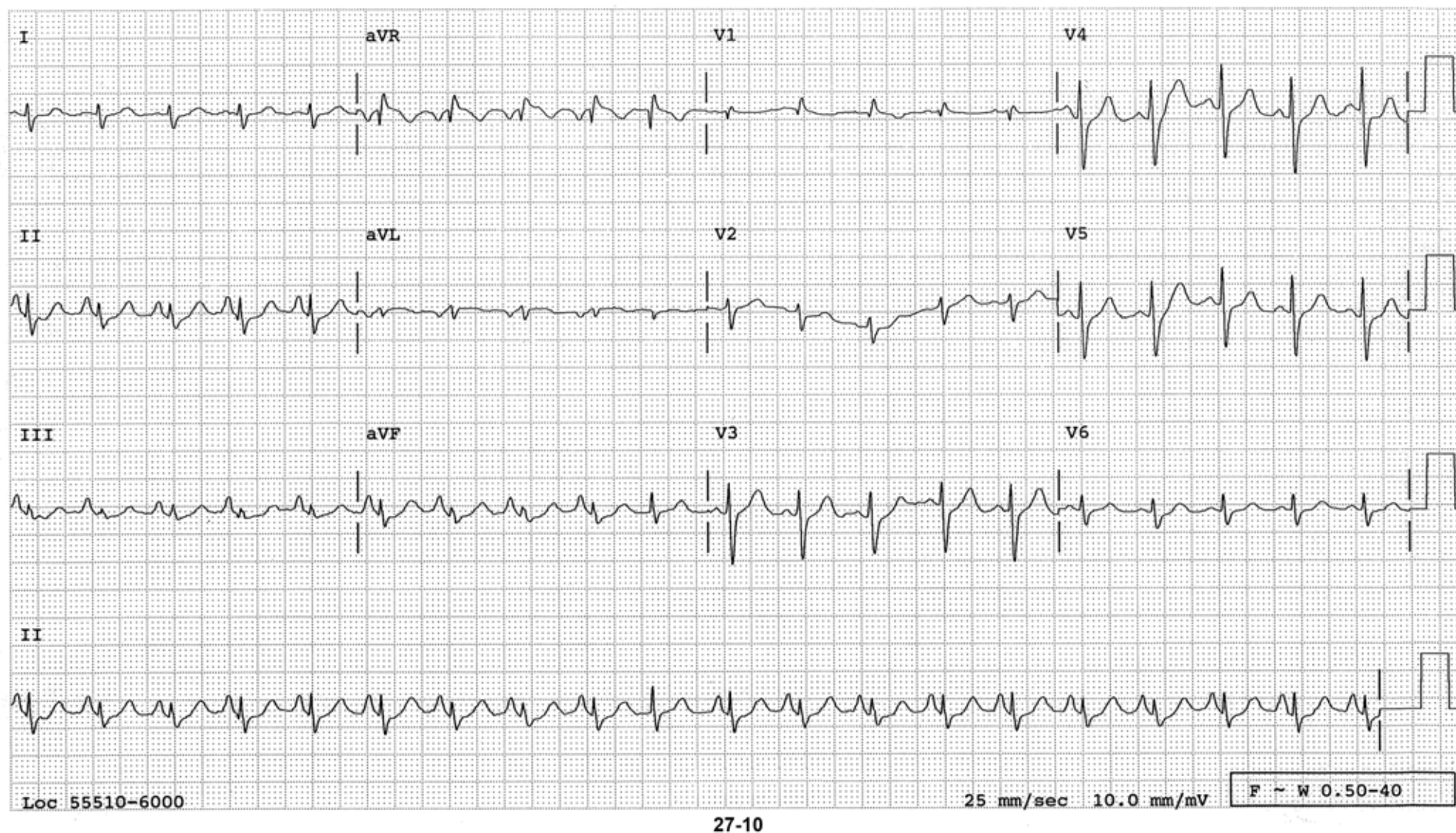
27-8 At first glance, the tracing raises the possibility of various rhythm disturbances such as atrial tachycardia, atrial flutter, MAT, etc. However, it is clear from the precordial leads that the patient is in NSR and that all the extra blips are artifacts. Poor R wave progression in the right precordial leads raises the possibility of old AMI.

Dx: Artifact simulating various rhythm disturbances



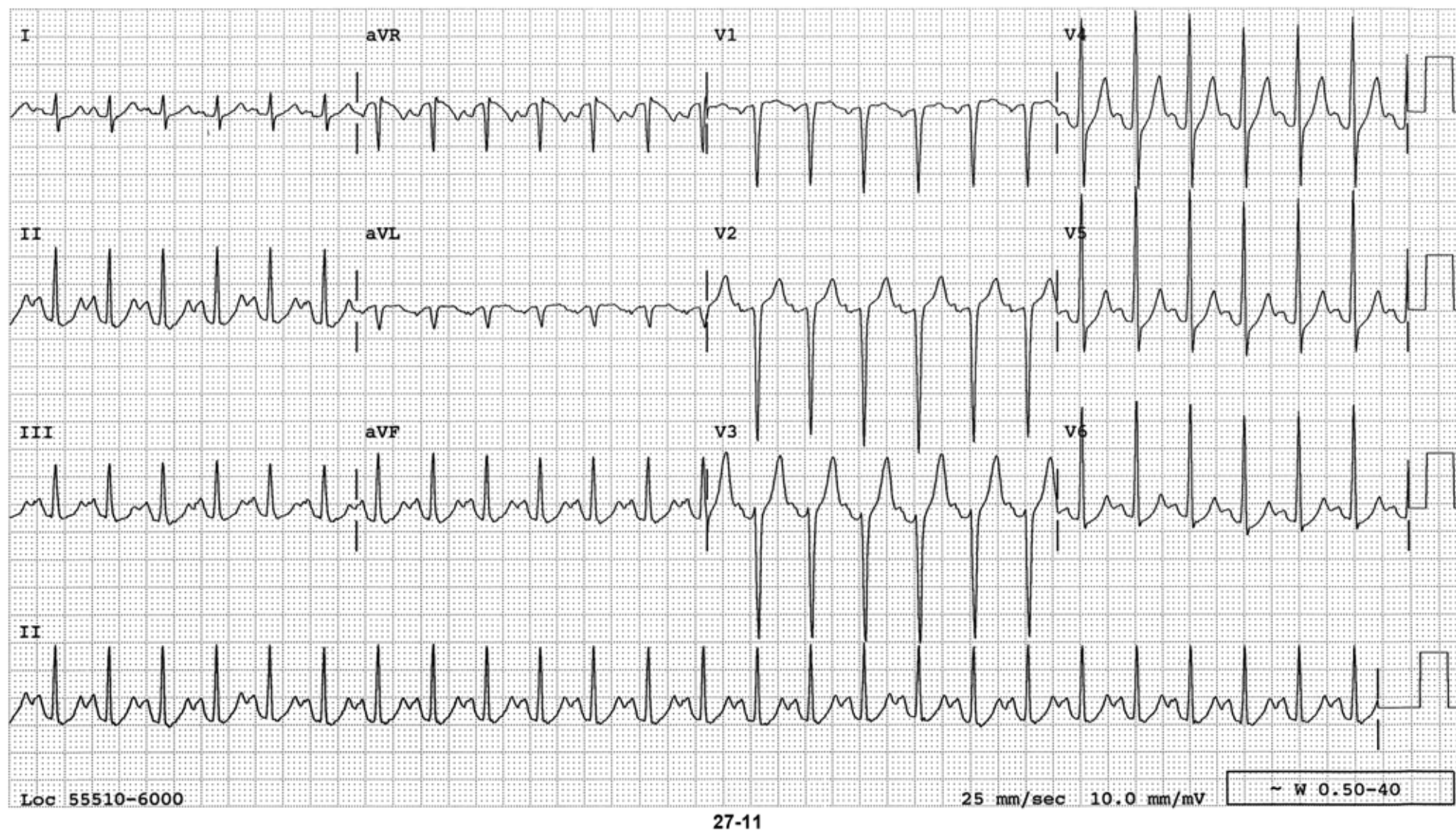
27-9 Narrow QRS tachycardia at a rate of 193/minute. No P waves can be definitively identified. This is a good example of SVT. Mild ST depression in the left precordial leads may or may not be due to myocardial ischemia because tachycardia alone without ischemia is well-known to cause ST depression.

- Dx: 1. SVT
2. ST-T changes of unknown significance



27-10 Sinus tachycardia at a rate of 117/minute. RAD, tall R waves in V_1 and deep S waves in V_6 are all diagnostic features of RVH. The P waves are tall, measuring 3 mm in the inferior leads, indicating RAE as well. The QRS voltage is small. This RVH may be 2° to COPD

- Dx:*
1. Sinus tachycardia
 2. RAE
 3. RVH, probably 2° to COPD



27-11 Sinus tachycardia at 150/minute. In the inferior leads and V_4 - V_6 the PR-segment is smoothly downsloping. The smoothly upsloping ST-segment begins at the same level as the end of the PR-segment so that the PR and ST-segments make a smooth, scooped out curvature. These are atrial repolarization waves (Ta wave). Ordinarily atrial repolarization does not cause much of wave but occasionally, especially during sinus tachycardia or stress test, the Ta wave becomes pronounced as in this case. It is important to recognize this prominent Ta wave so that one does not think of pathologic PR-segment depression seen in pericarditis or atrial infarction or pathologic ST depression.

- Dx: 1. Sinus tachycardia
2. Prominent Ta wave, a normal phenomenon

27-12 Question: This rhythm strip reveals (choose one from below):

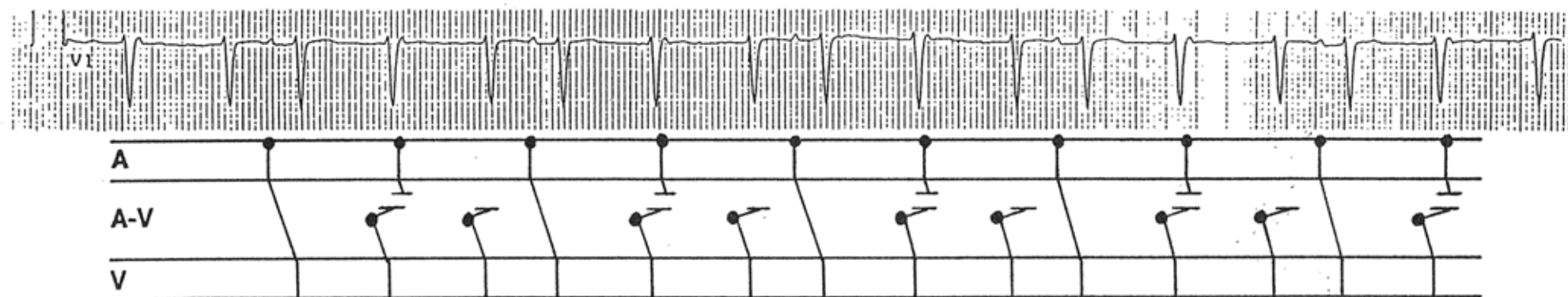
- (A) AV junctional acceleration with occasional premature atrial beats
- (B) NSR and AV junctional acceleration with AV dissociation and occasional capture beats
- (C) Extreme sinus bradycardia (rate 34/minute) and AV junctional acceleration and occasional capture beats
- (D) Extreme sinus bradycardia and 3° AV block with AV junctional escape rhythm



27-12A

Answer: (B) Normal sinus rhythm with AV junctional acceleration with AV dissociation and occasional capture beats as diagrammed in Figure 27.12B

Discussion: As diagrammed, the atria are driven by the sinus impulse at a rate of about 65/minute. The primary problem in this patient is AV junctional acceleration. Physiological refractoriness of the conduction system results in AV dissociation. When the P wave occurs at the right time, it will conduct to the ventricle, resulting in a capture beat. The hallmark of the capture beat is that the QRS occurs with a shorter R-R interval than other beats. In this situation, one has to look for the cause of the junctional acceleration, which is the primary problem, while AV dissociation and capture beats are all obligatory secondary phenomena. Digitalis toxicity, myocardial ischemia or infarction, or increased sympathetic activity is well-known causes of AV junctional acceleration.

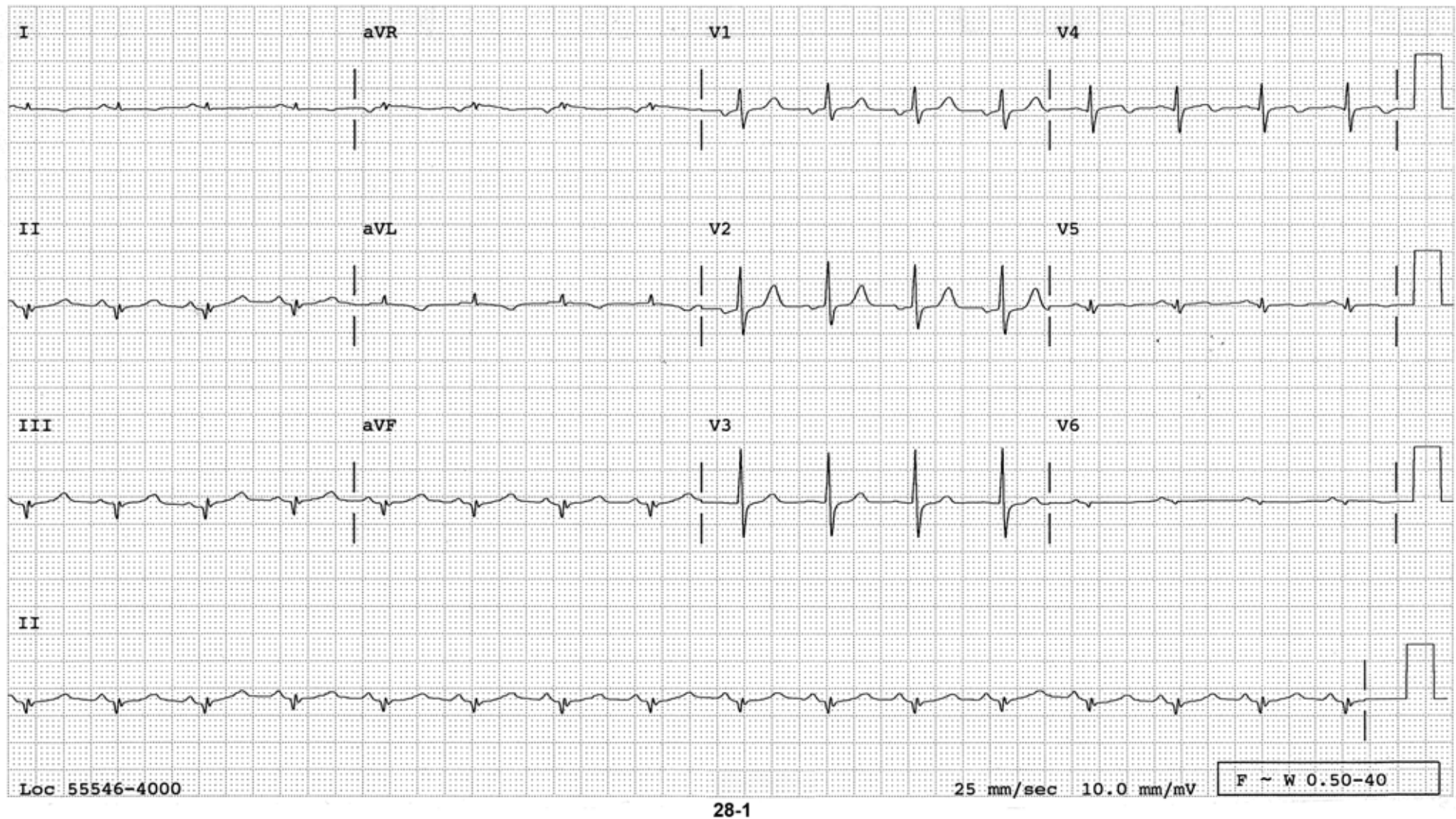


27-12B

SECTION 28

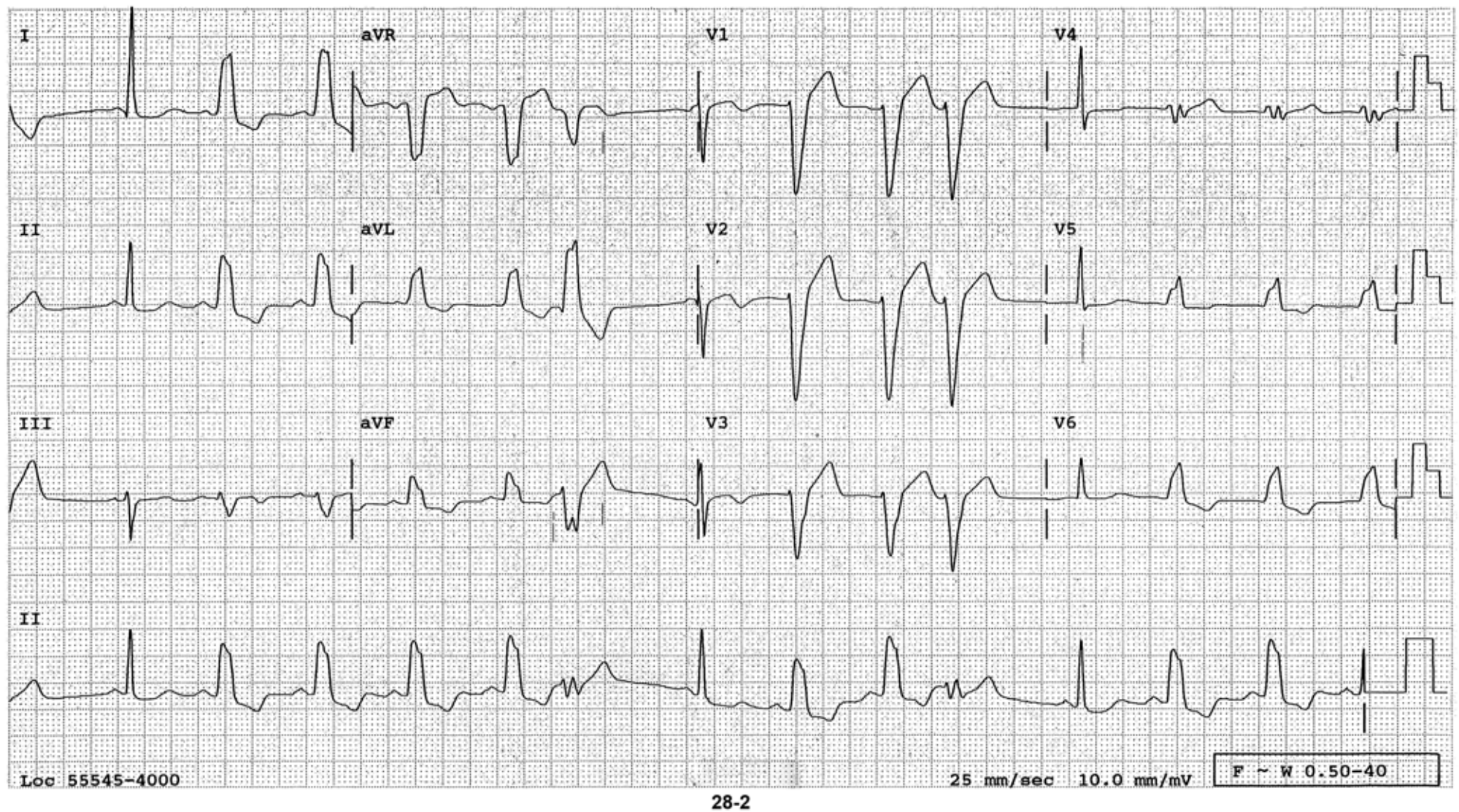
28-1 Question: R waves are tall in the right precordial leads. This tracing reveals (choose one below):

- (A) RVH
- (B) COPD
- (C) Inferoposterolateral MI
- (D) WPW syndrome



Answer: (C) Inferoposterolateral MI

Discussion: Q waves in the inferior leads and QS pattern in V_6 are helpful clues that the tall R waves in the right precordial leads are due to inferoposterolateral MI. The P-R interval is not short in any of the twelve leads, and there are no convincing delta waves, ruling out WPW syndrome. COPD causes either poor R wave progression or even QS pattern in the right precordial leads rather than tall R waves in these leads. RVH should be associated with RAD which is not the case here.



28-2 Normal sinus rhythm at a rate of 85/minute. Most ORSs have LBBB pattern. Occasionally, the QRS normalizes. After the pause following PVC, the left bundle branch has more time to recover and conducts normally. This proves that the LBBB in this patient is not constant, but is rate dependent. Sometimes, this normally conducted QRS may reveal very useful information which is masked by LBBB. Another example of usefulness of PVCs:

- Dx:
1. NSR
 2. Rate-dependent LBBB
 3. Frequent PVCs

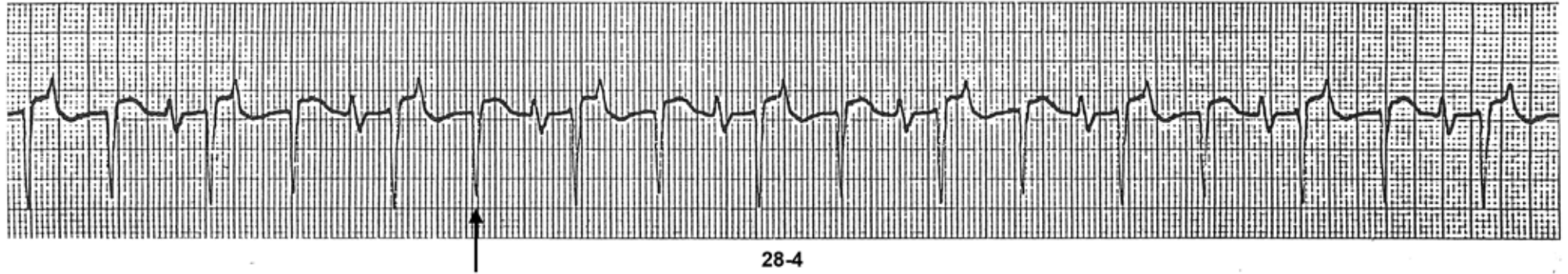


28-3 Sinus bradycardia at 47/minute. T waves are tall in V_2 - V_4 but are asymmetric (the upstroke takes a longer time than the downstroke). These are tall T waves of normal variant. Tall T waves of hyperkalemia or hyperacute T wave changes are symmetric.

- Dx: 1. Sinus bradycardia
2. Tall T waves of normal variant

28-4 Question: The QRS complex pointed by an arrow is (choose one from below):

- (A) An atrial premature beat with a long P-R interval
- (B) An AV junctional beat following a nonconducted PAC
- (C) An AV junctional premature beat as a part of AV junctional bigeminy



Answer: (A) An atrial premature beat with a long P-R interval

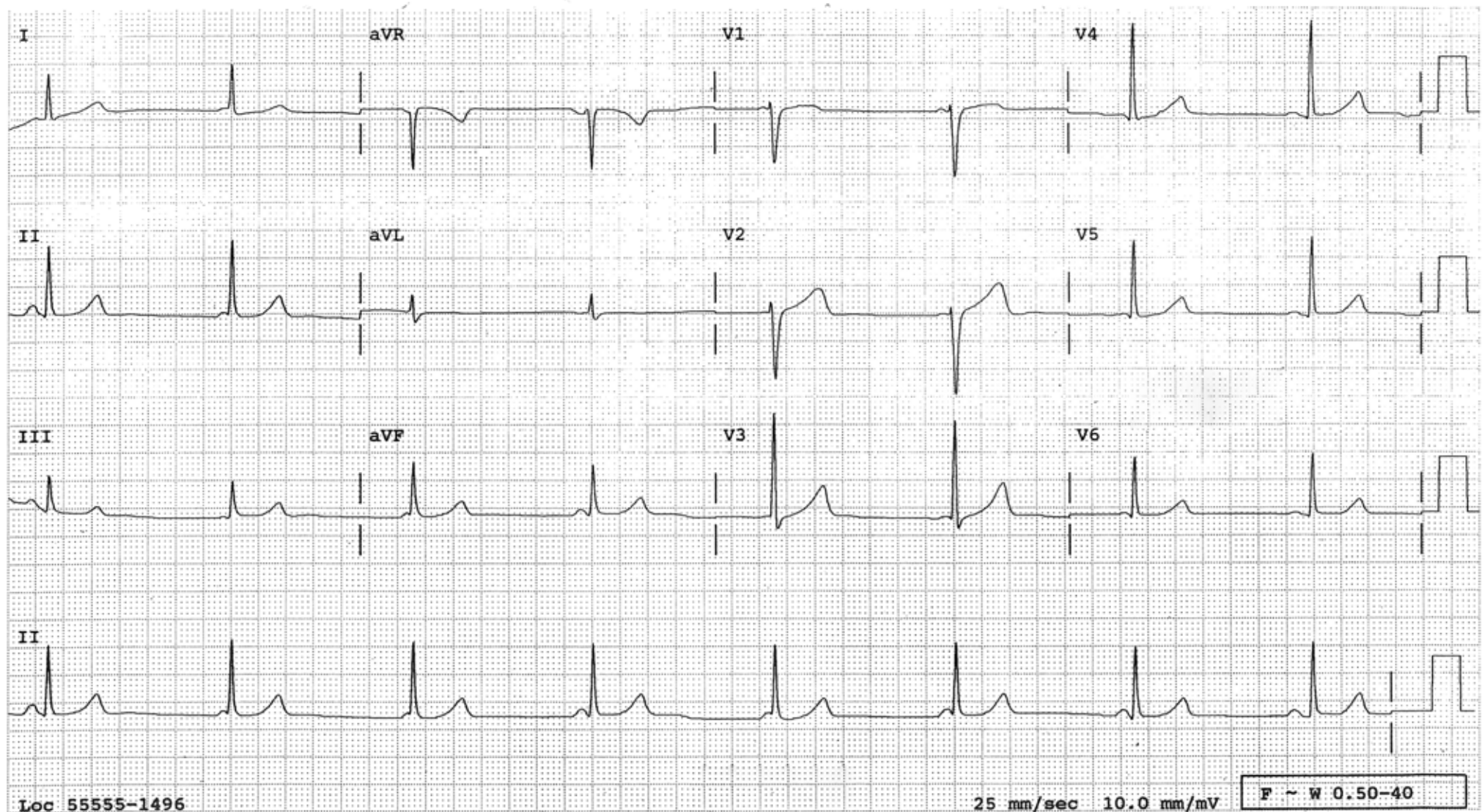
Discussion: The T wave of the QRS in front of the one pointed by an arrow is very pointed due to a premature P wave superimposed on it. This P wave occurs during the relative refractory period of the AV conduction system, resulting in a long P-R interval.

28-5 Question: What clinical condition should this 12 lead ECG tracing make you think of?



Answer: Hypothermia

Discussion: Slurred downstroke of the QRS is evident in many leads including the inferior leads as well as the precordial leads. These are Osborn waves (J waves) characteristic of hypothermia. Atrial fibrillation is a common rhythm disorder in hypothermia.



28-6

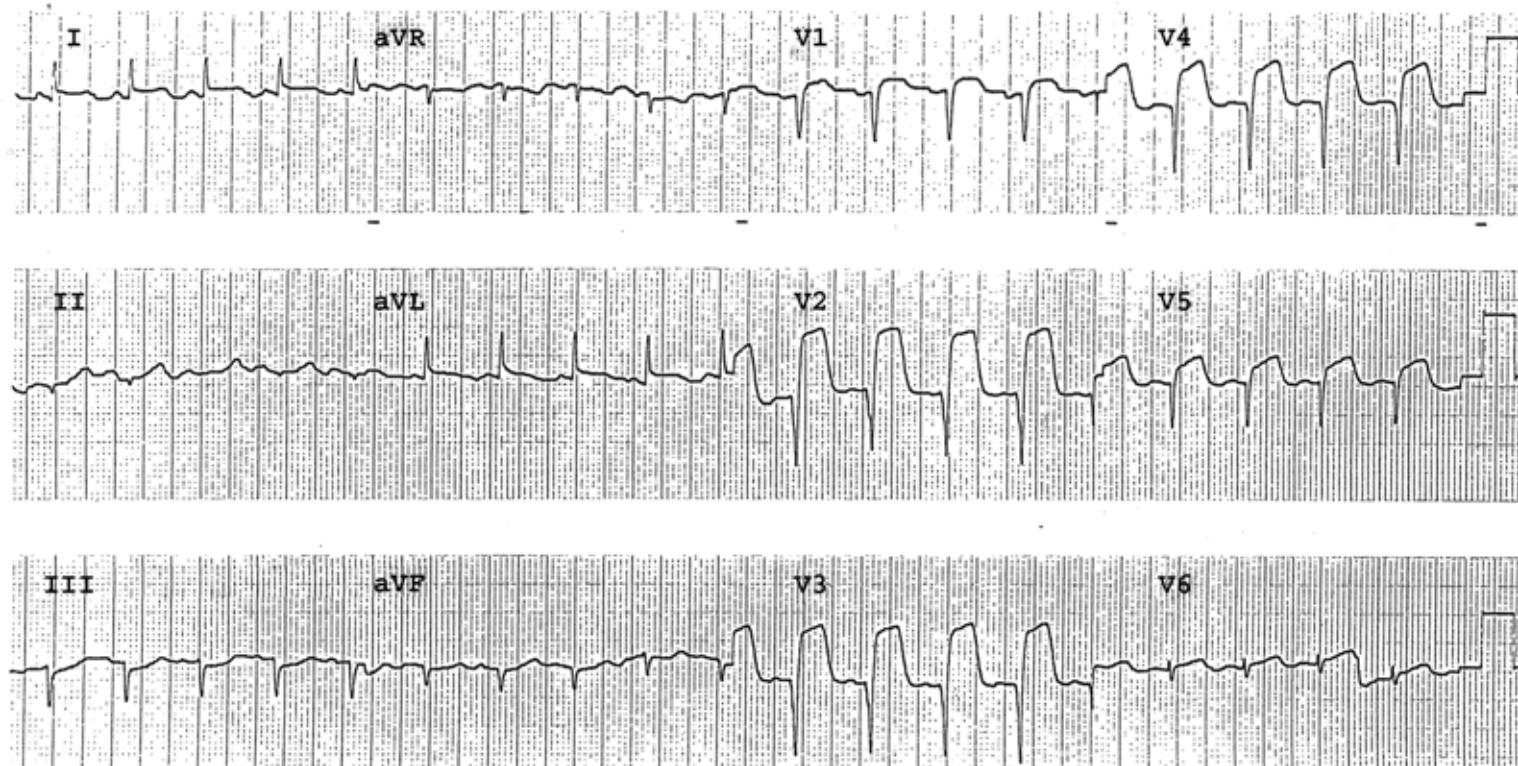
28-6 The P waves occur regularly at a rate of about 43/minute. This sinus bradycardia allows the AV junctional pacemaker to escape at just about the same rate, resulting in AV dissociation. The primary disorder here is sinus bradycardia, and the AV junctional escape is merely a physiologic response to the primary disorder. AV dissociation in this case does not imply AV block but is merely a reflection of physiologic refractoriness of the conduction system.

Dx: Sinus bradycardia with AV junctional escape rhythm and AV dissociation

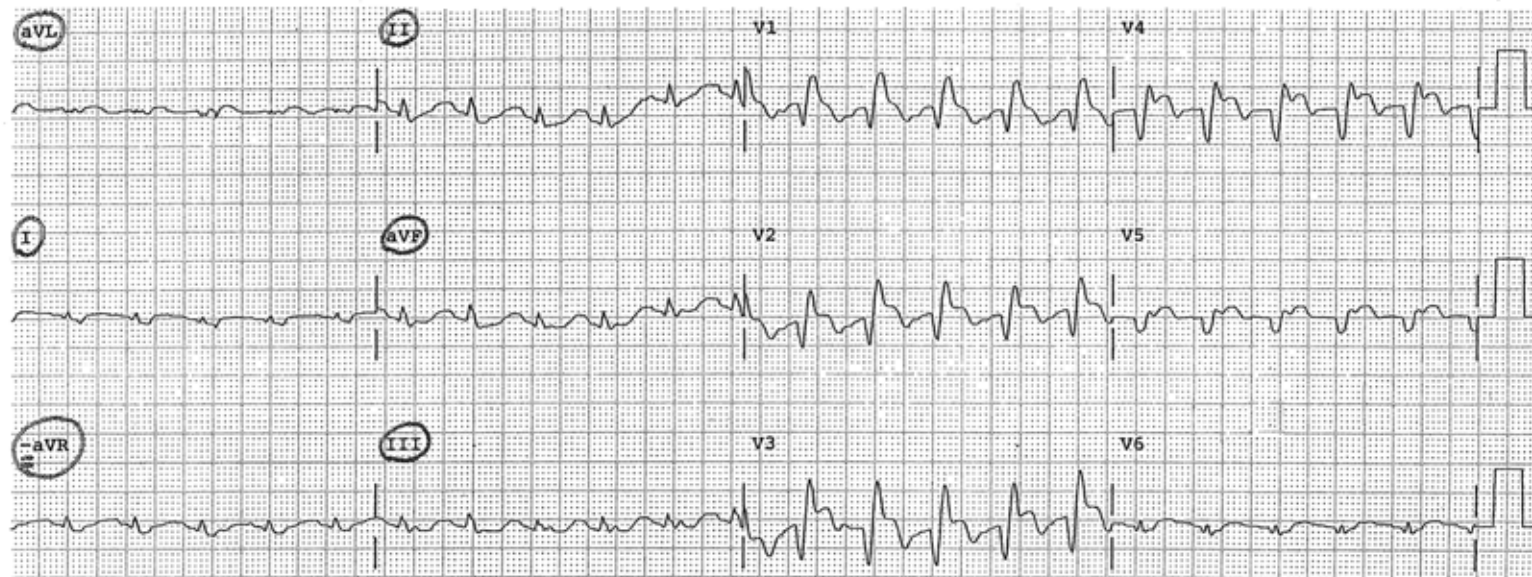
28-7 Question: An acute MI complicated by a new BBB carries an increased inhospital mortality. Which of the tracings below reveal(s) an acute anteroseptal MI and RBBB?

- (A) Patient A
- (B) Patient B
- (C) Both
- (D) Neither

Patient A



Patient B



28-7

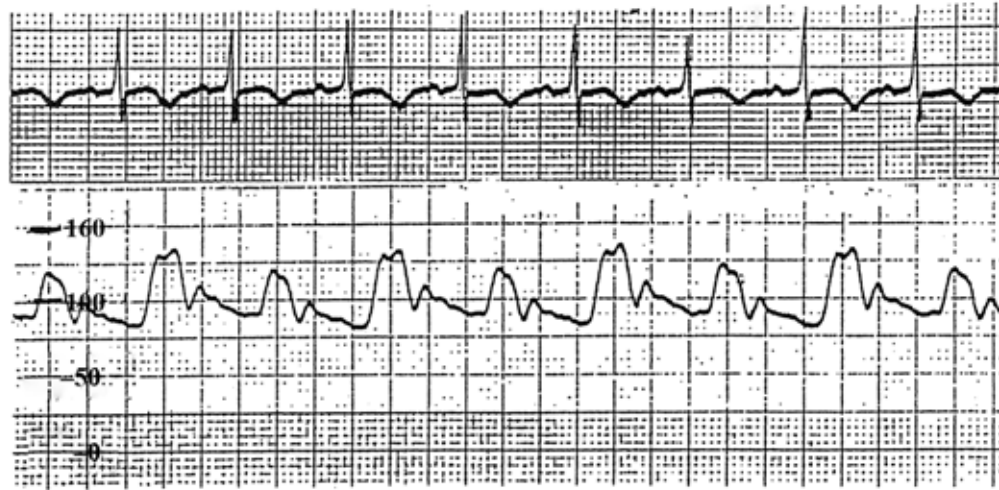
Answer: (B) Patient B

Discussion: The right precordial leads of patient B have the second "rabbit ear" of RBBB sticking out while those of patient A have smooth ST-segment elevation only.

28-8 *Question:* In pulsus alternans (= mechanical alternans), strong and weak pulses alternate and signifies severe ventricular dysfunction. Following figures are arterial pressure tracings taken simultaneously with ECG from two different patients. Which of the following patients reveal(s) pulsus alternans?

- (A) Patient A
- (B) Patient B
- (C) Both
- (D) Neither

Patient A



Patient B



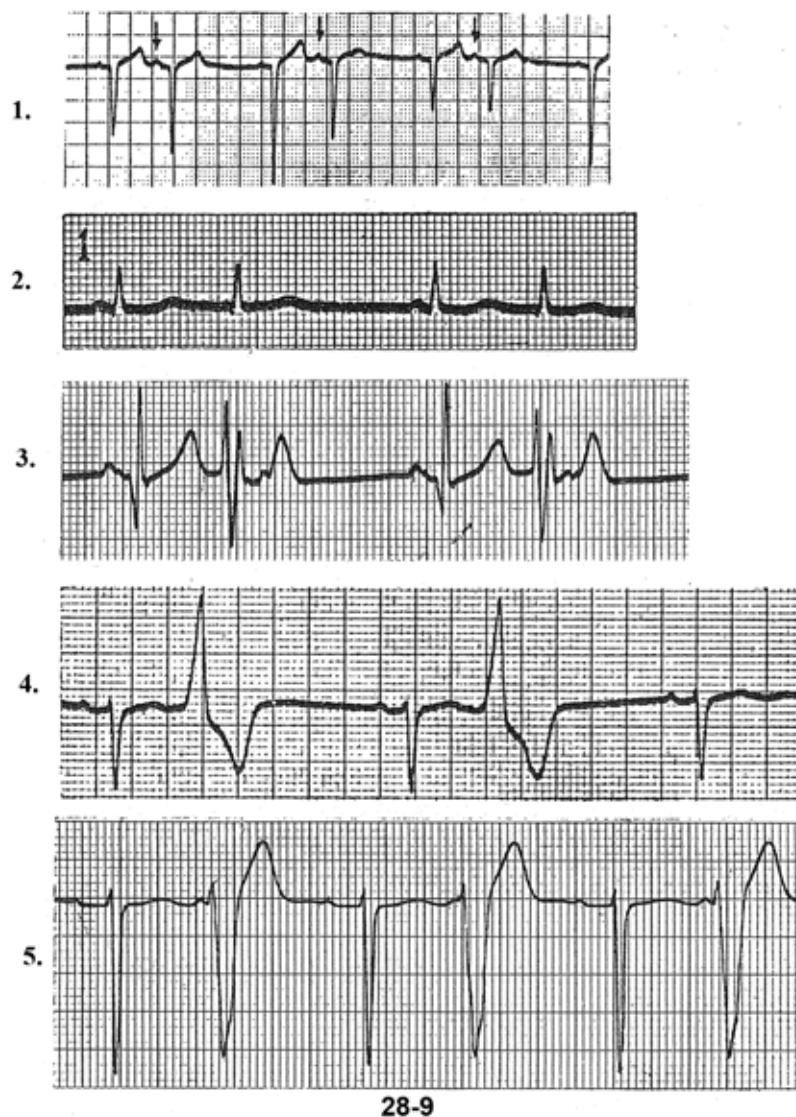
28-8

Answer: (A) Patient A

Discussion: The tracing of patient B also reveals strong and weak pulse alternating. However, it is due to the alternating diastolic filling period secondary to bigeminal rhythm. Pulsus alternans is a situation where a strong and weak pulsus alternate during a regular rhythm as seen in patient A. The pulse in patient B is called bigeminal pulse (pulsus bigeminus), which is due to alternating diastolic filling time, a manifestation of Frank-Starling's law.

28-9 *Question:* What are the causes of bigeminal rhythm in these tracings? Match the tracings with the diagnoses listed below. The same diagnosis can be used more than once.

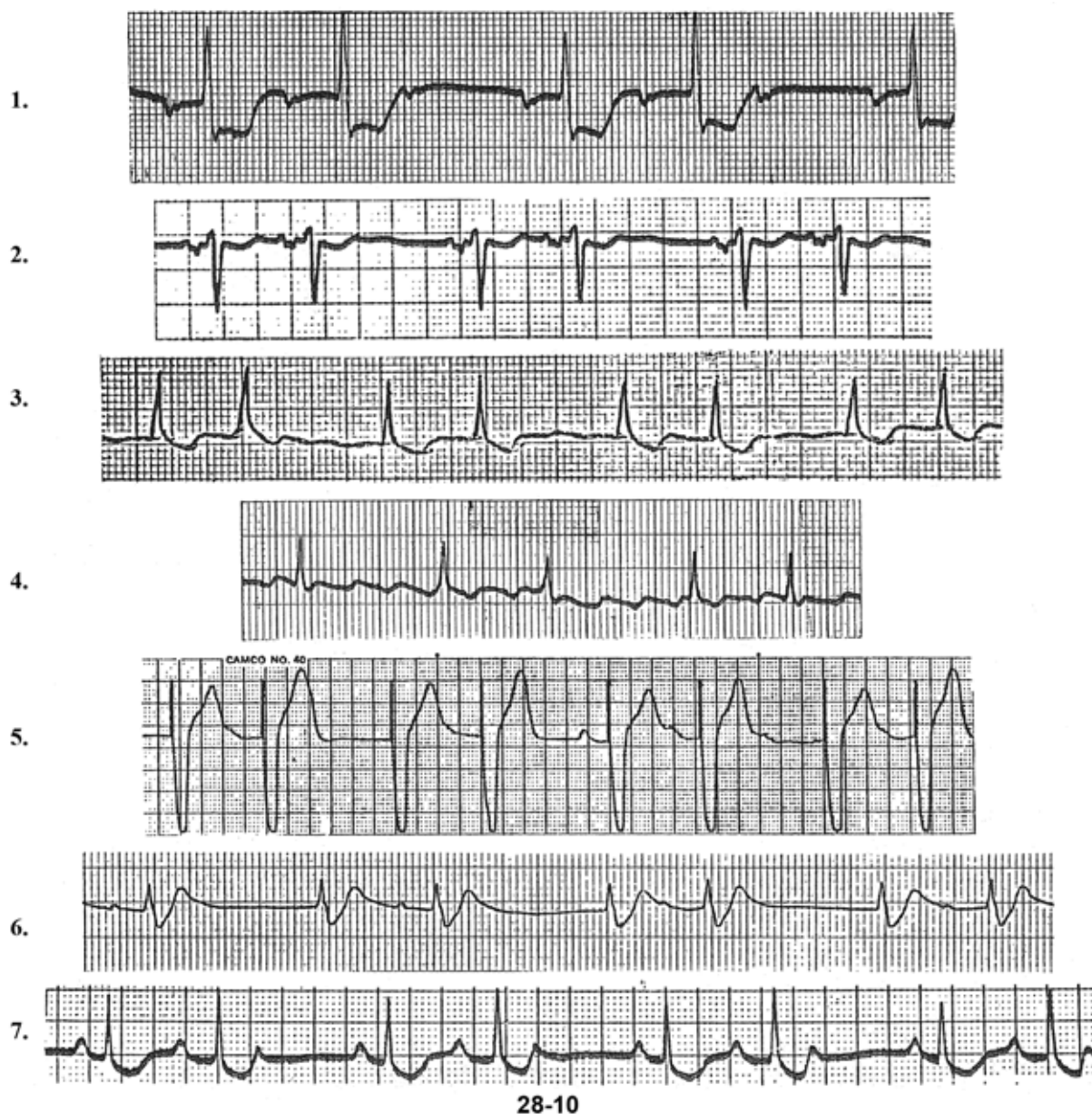
- (A) Atrial bigeminy
- (B) Junctional bigeminy
- (C) Ventricular bigeminy



28-9

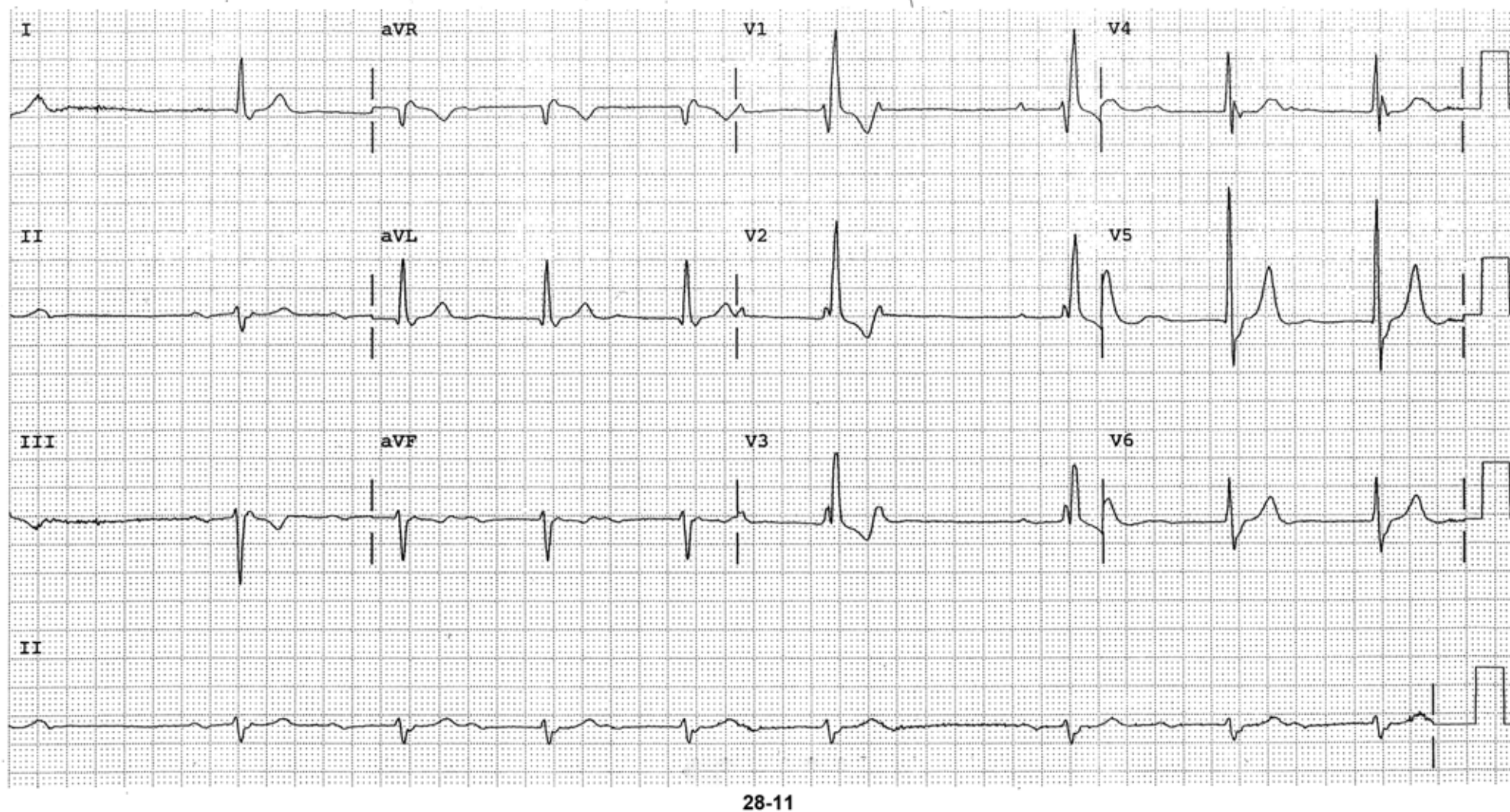
Answer:

1. (A) Atrial bigeminy in that every other QRS is preceded by a prematurely occurring P wave.
2. (B) Junctional bigeminy in that the QRSs have identical configuration and the second QRS of the pair is not preceded by a P wave.
3. (C) Ventricular bigeminy. The uninterrupted sinus P wave can be seen between the premature QRS and the T wave.
4. (C) Ventricular bigeminy. There is a P wave hidden within the premature QRS or its ST-segment and is not visible.
5. (C) Ventricular bigeminy where the PVCs occur late enough so that the sinus P wave is present in front of the PVC (barely visible).



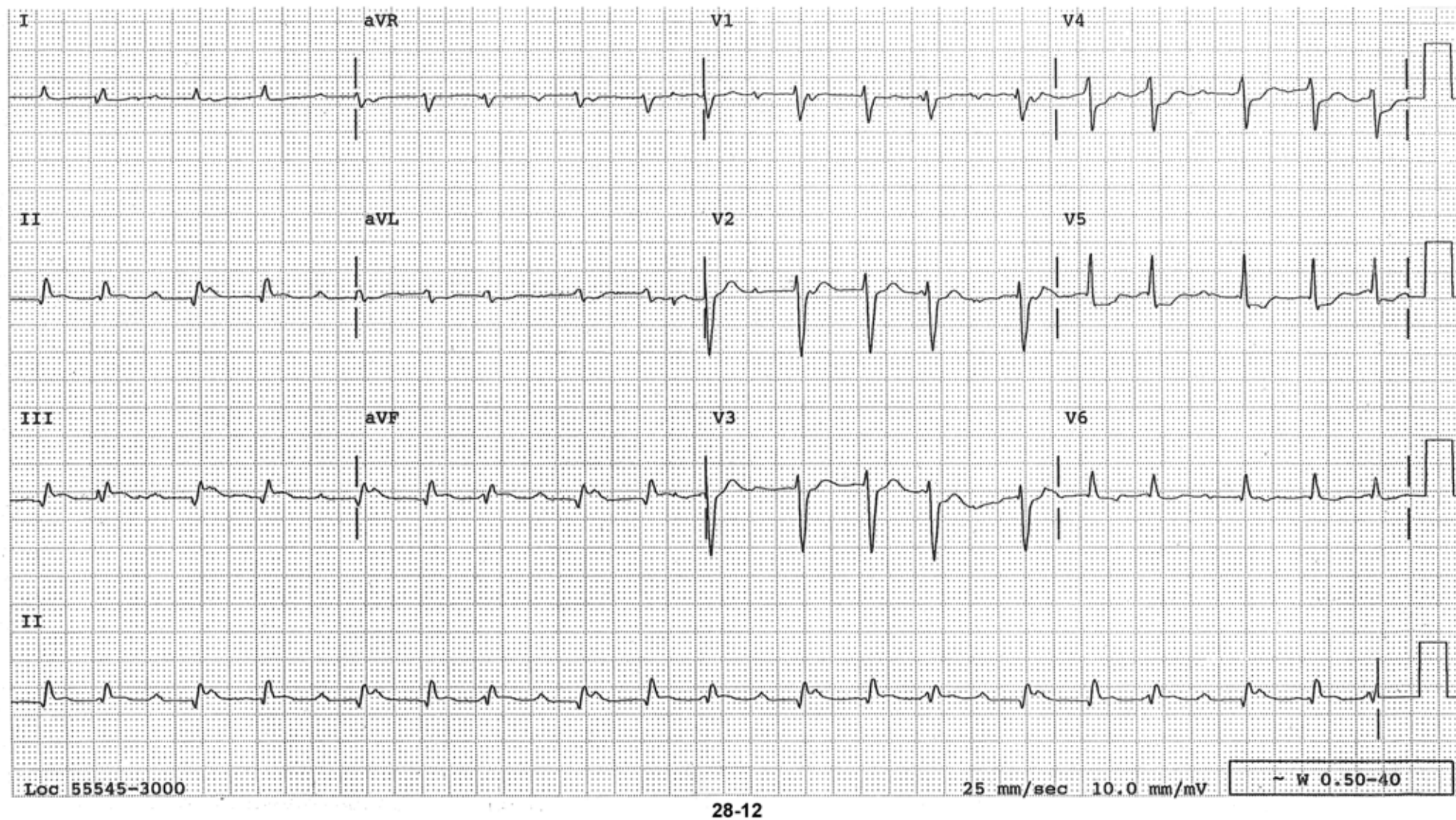
Answer:

1. 3:2 AV Wenckebach phenomenon. P waves occur regularly. Progressively lengthening P-R intervals with every third P wave being blocked is evident.
2. 3:2 SA block, Type I. There are no blocked P waves. The P-R interval stays the same. Every third sinus impulse is blocked, resulting in no P wave and no QRS. The longer R-R interval is less than two shorter R-R intervals, making this Type I.
3. Junctional tachycardia with 3:2 exit block. The patient is in atrial fibrillation. The ventricle is driven by AV junctional tachycardia, but every third impulse fails to propagate to the ventricle and every second QRS is slightly delayed. That is why the longer R-R interval is shorter than two shorter R-R intervals. Digitalis toxicity should be strongly considered.
4. Atrial flutter with variable AV conduction ratio. Alternating 4:1 and 3:1 AV conduction will result in bigeminal rhythm.
5. "Artificial pacemaker bigeminy". This demand ventricular pacemaker is sensing the peak of every other T wave as a QRS and it does not fire until the programmed pacing interval from that point, resulting in bigeminy (pacemaker oversensing). Note that the sensed T waves are taller than the other T waves.
6. Extreme sinus bradycardia with AV junctional escape complexes and retrograde block from the AV node to the atrium which allows noninterruption of the sinus mechanism. Were it not for the retrograde block to the atria, it would have been AV junctional rhythm at a regular rate with 1:1 retrograde conduction to the atria. The primary disorder in this case is extreme sinus bradycardia and retrograde VA block.
7. Bigeminy secondary to nonconducted atrial trigeminy. Every other T wave is pointed and this is due to a premature P wave superimposed on top of the T wave. This premature P wave is blocked (nonconducted atrial trigeminy).



28-11 P waves occur regularly. P-R interval lengthens until a P wave is blocked; a typical AV Wenckebach phenomenon. RBBB and LAFB constitute BIFB.

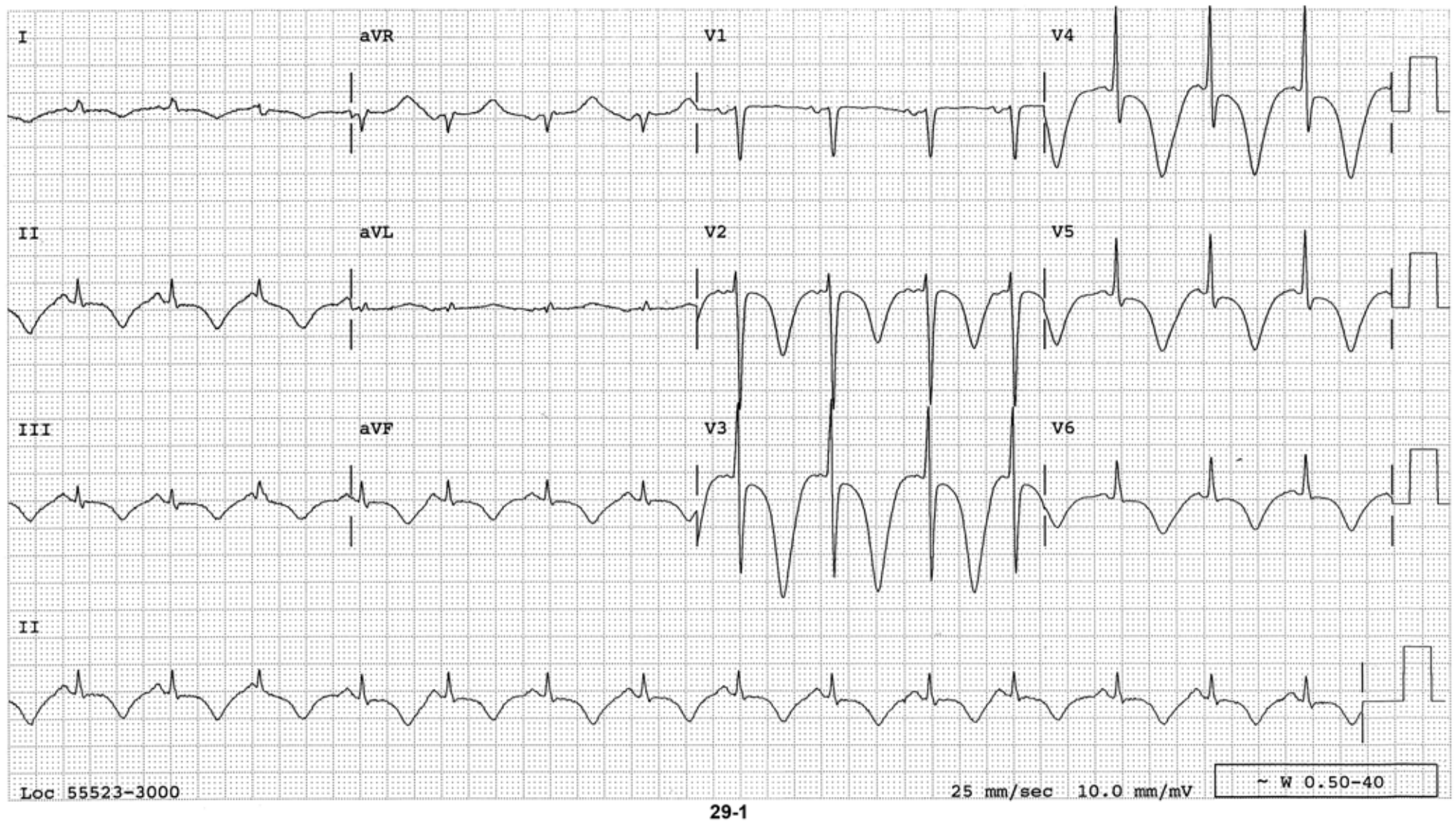
- Dx: 1. Sinus rhythm with AV Wenckebach phenomenon
2. Bifascicular block (RBBB + LAFB)



28-12 There is a group beating of QRSs. P waves occur regularly at a rate of about 150/minute. Some P waves occur within the QRS. Progressive lengthening of the P-R interval can be appreciated. Third or fourth P waves are blocked: typical AV Wenckebach phenomenon. QRS voltage is abnormally low in the limb leads. Q wave with ST elevation in inferior leads is diagnostic of inferior STEMI, which may be the cause of this AV block.

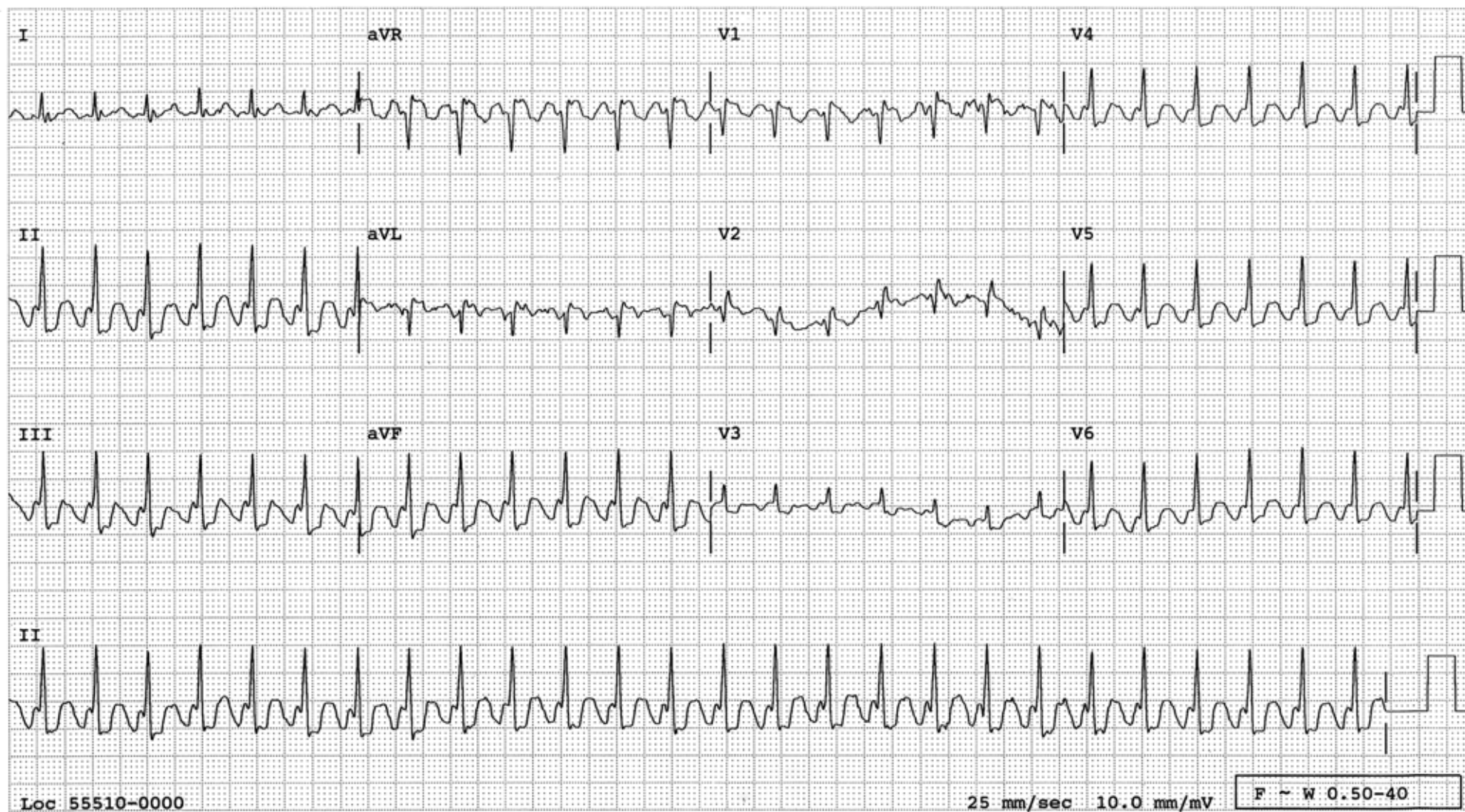
- Dx:*
1. Sinus tachycardia
 2. AV Wenckebach phenomenon
 3. Inferior STEMI
 4. Abnormally low QRS voltage in the limb leads

SECTION 29



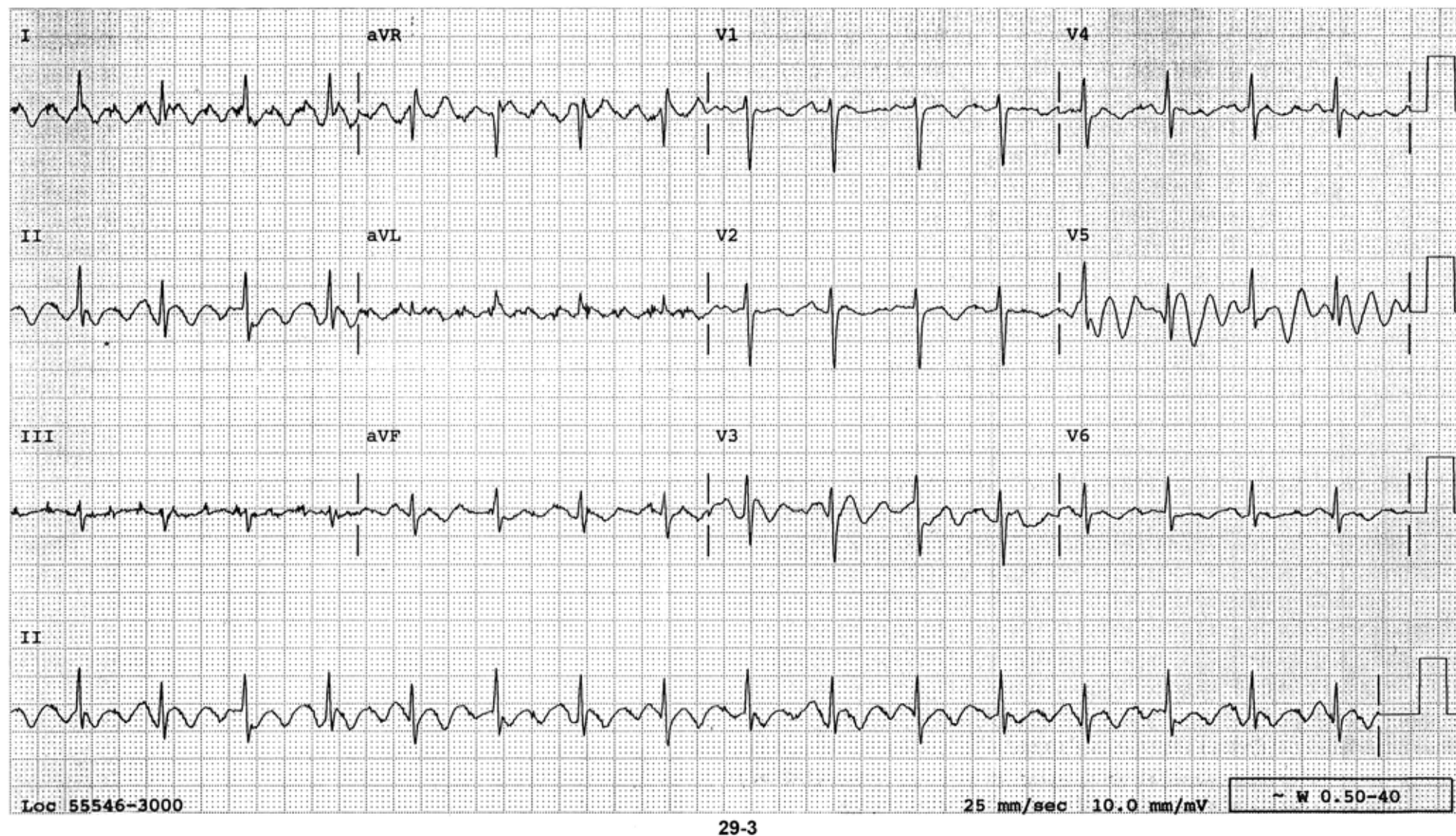
29-1 Normal sinus rhythm at 87/minute. Deeply and symmetrically inverted T waves combined with long Q-T interval are the ECG findings seen in the so-called stress cardiomyopathy (also called apical ballooning syndrome, stunned myocardium, broken heart syndrome, etc.) due to a variety of conditions, such as CNS events or other causes of catecholamine surge.

- Dx: 1. NSR
2. Findings highly suggestive of stress cardiomyopathy



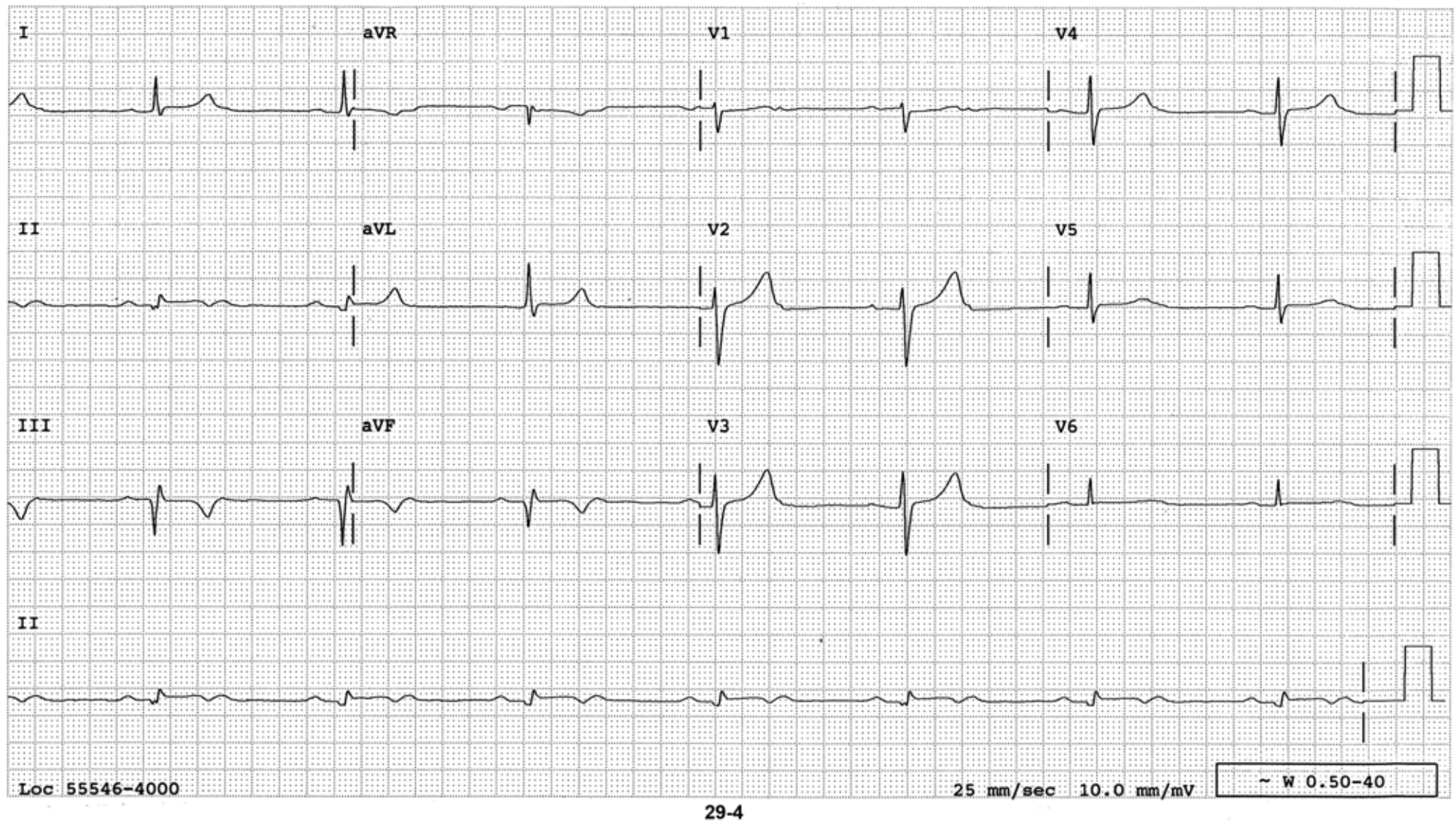
29-2 A typical example of atrial flutter with 2:1 AV conduction. ST-segment depression in the inferolateral leads may or may not be secondary to myocardial ischemia.

- Dx:*
1. Atrial flutter with 2:1 AV conduction
 2. ST-T changes may or may not be due to myocardial ischemia



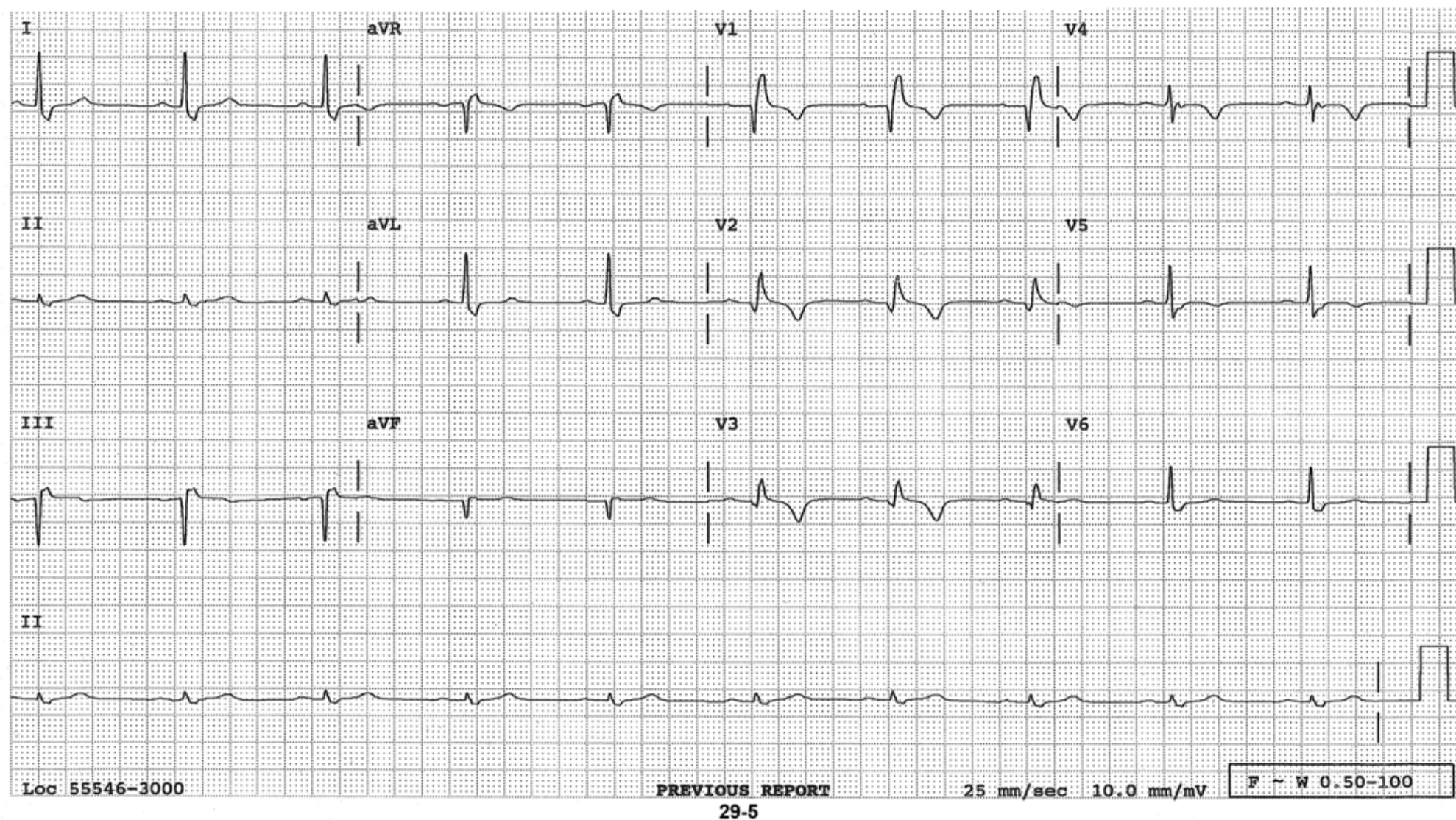
29-3 An example of artifact from muscle tremor in Parkinson's disease mimicking atrial flutter. Sinus P waves can be appreciated in V_1 , V_2 , V_4 , and V_6 . Besides, the QRS complexes occur regularly which means, if this were atrial flutter there should be a fixed AV conduction ratio. In that case, the QRS complexes and the flutter waves should maintain a fixed relationship, which is not happening here (see lead I). Also the flutter waves are in the wrong lead. They should be seen primarily in the inferior leads, not in lead I.

Dx: Artifact mimicking atrial flutter



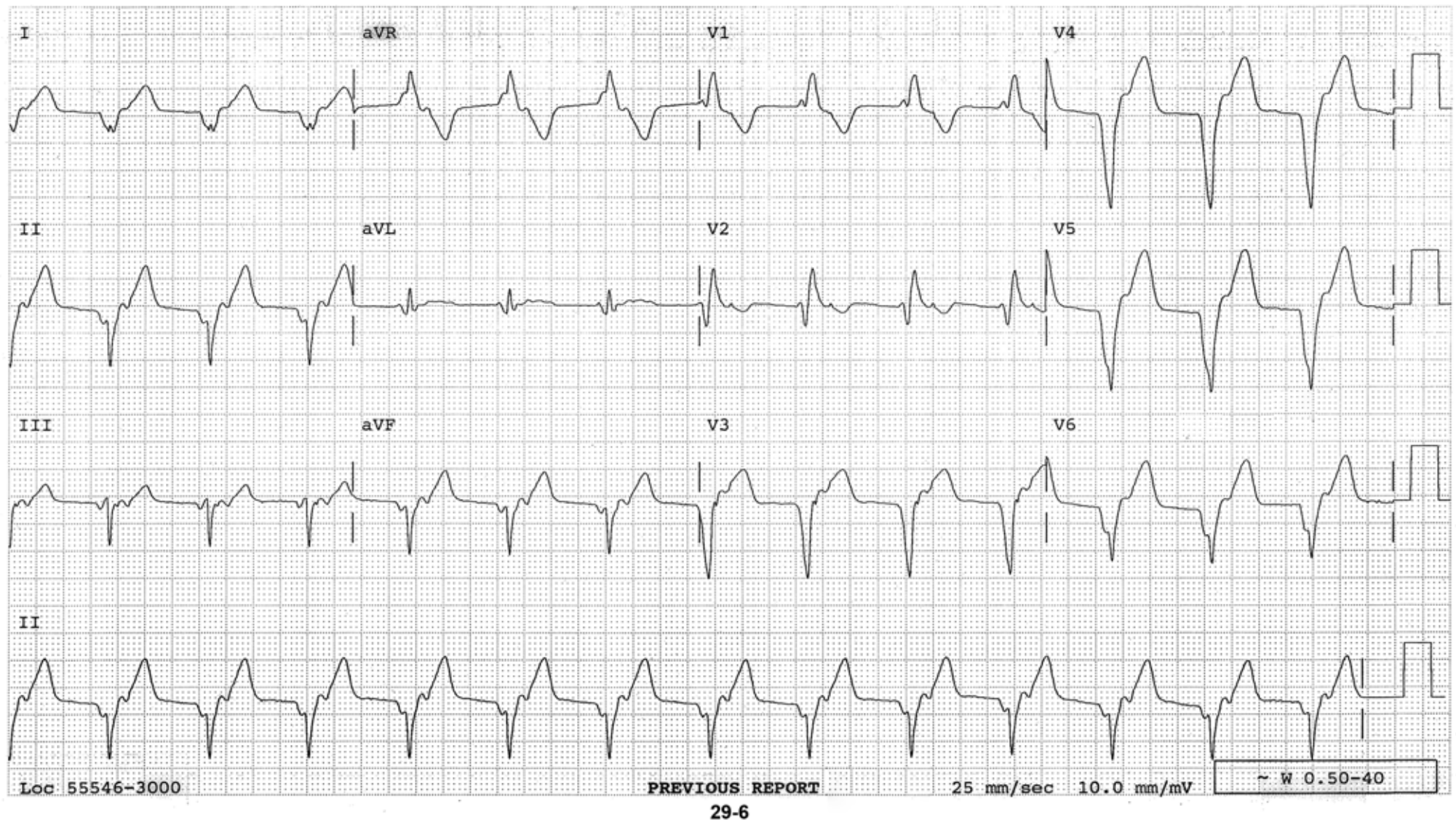
29-4 At first glance, the rhythm appears to be sinus bradycardia at a rate of about 43/minute. However, careful observation reveals an additional P wave between the QRSs, best appreciated in V_1 - V_3 , indicating sinus rhythm at a rate of 85/minute with 2:1 AV block. The QRSs are narrow and the conducted P-R interval is long, indicating the block is within the AV node. Q waves with T wave inversion in the inferior leads indicate recent inferior MI. The 2:1 AV block, most likely, is secondary to inferior MI, and in that case, the AV conduction disorder should improve.

- Dx: 1. NSR with 2:1 AV block
2. Acute inferior infarct



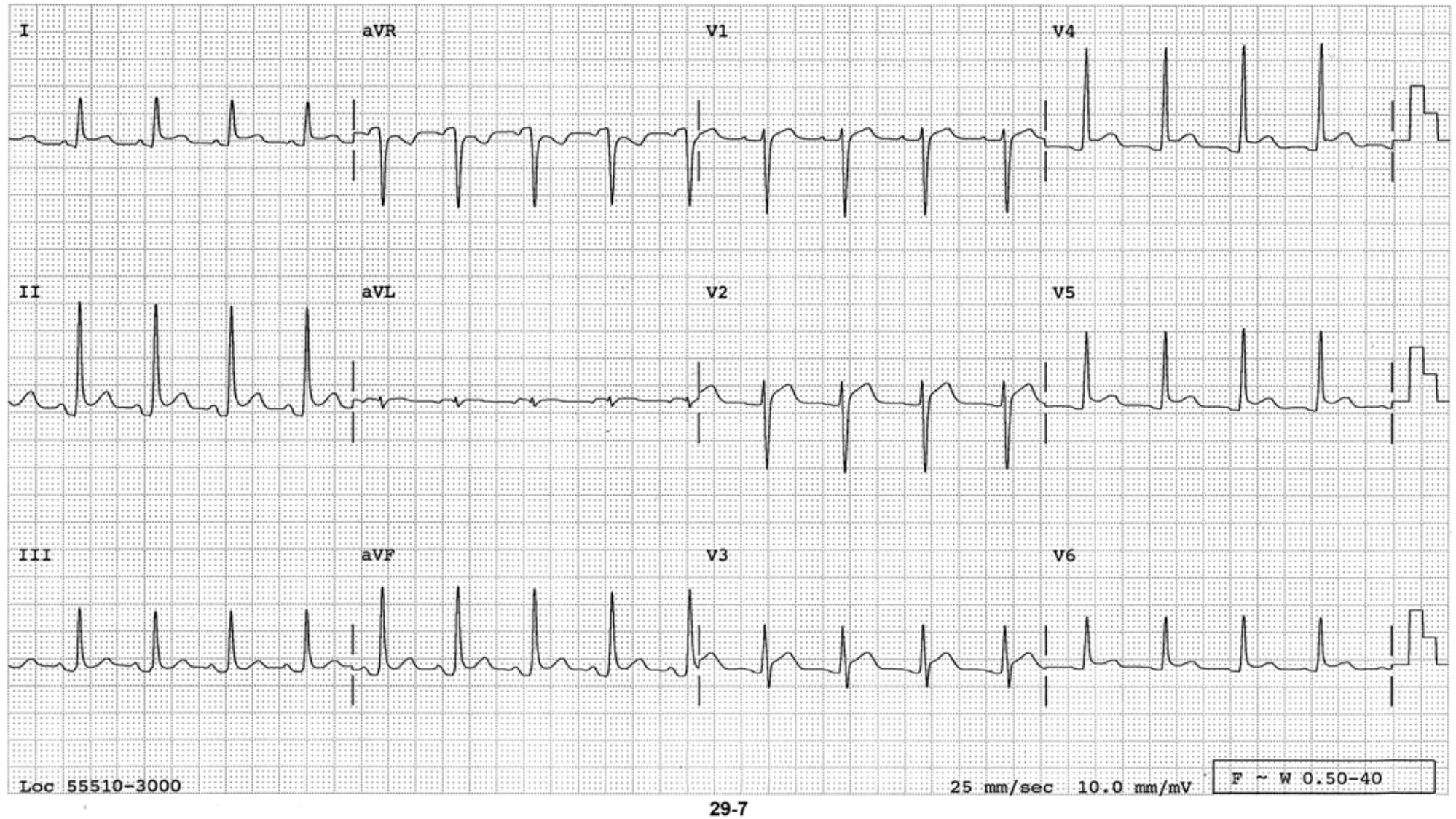
29-5 Normal sinus rhythm at a rate of 58/minute. Complete RBBB is present. Q waves in V_1 - V_3 indicate anteroseptal infarction. Q waves in leads III and aVF indicate old inferior infarction as well.

- Dx:
1. NSR
 2. RBBB
 3. Anteroseptal infarction, most likely recent
 4. Old inferior infarction



29-6 Regular wide QRS rhythm at a rate of 80/minute. No P waves are seen in front of the QRS. A negative deflection soon after the QRS, especially in the inferior leads and in V_3 , indicates a retrograde P wave. The tracing is an example of accelerated idioventricular rhythm with 1:1 VA conduction. Accelerated idioventricular rhythm is most commonly seen in the setting of an acute MI.

Dx: Accelerated idioventricular rhythm with 1:1 VA conduction

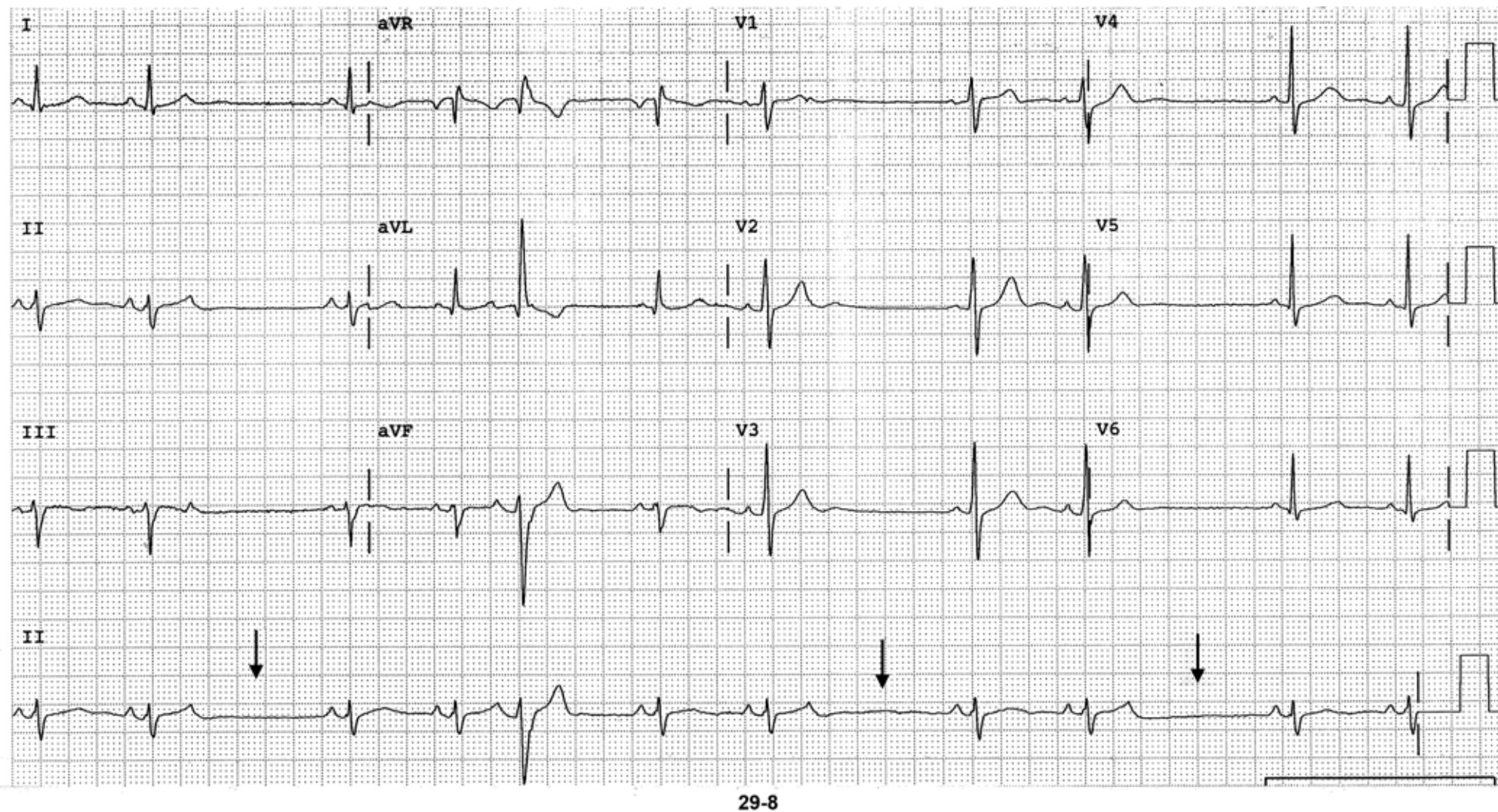


29-7 Sinus tachycardia at a rate of 110/minute. The ST-segment is elevated diffusely. The PR-segment is depressed, especially noticeable in the inferior leads. These findings are all consistent with pericarditis.

- Dx: 1. Sinus tachycardia
2. Acute pericarditis

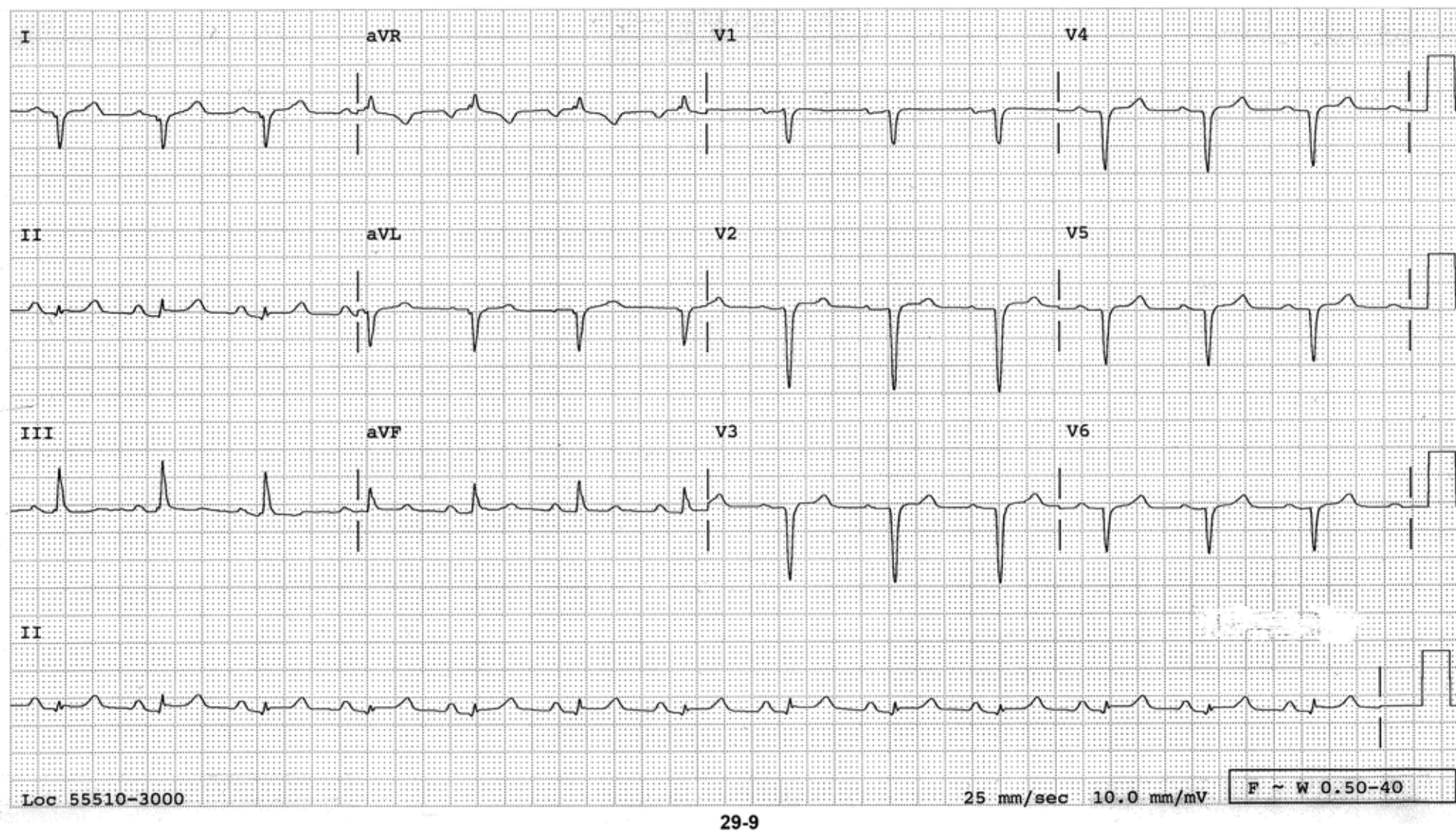
29-8 Question: What are the pauses (↓) due to: (choose one from below):

- (A) Sinus arrhythmia
- (B) SA block
- (C) Nonconducted PACs
- (D) 2° AV block



Answer: (C) Nonconducted PACs

Clues: The T wave at the beginning of the pauses is pointed and different from other T waves indicating that it is distorted by something such as a P wave. This is proven by a conducted PAC (fifth QRS) albeit it is conducted aberrantly.

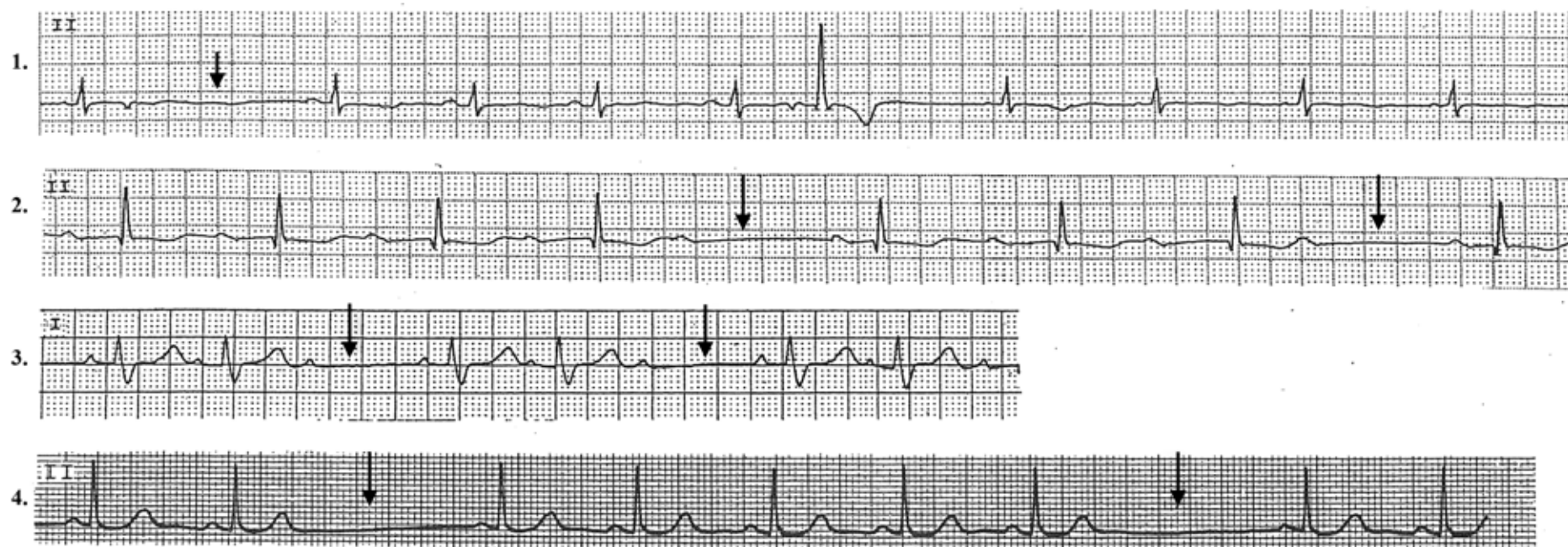


29-9 Normal sinus rhythm at a rate of 75/minute. The axis is shifted markedly to the right. RAD should make one think first of RVH; however, this is an example of RAD due to extensive anterior infarct (no R waves in the entire precordial leads). The positive P wave in lead I rules out the possibility of reversed arm leads.

- Dx:
1. NSR
 2. RAD
 3. Extensive old anterior infarct

29-10 Question: What is the cause of the pause in each of the following tracings?

Tracings 1–4 are rhythm strips from four different patients. They all have pauses (↓).



29-10

- Answer:
1. Nonconducted atrial premature impulse
 2. 2° AV block, Type I
 3. 2° AV block, Type II
 4. Sinoatrial block, Type II

Discussion:

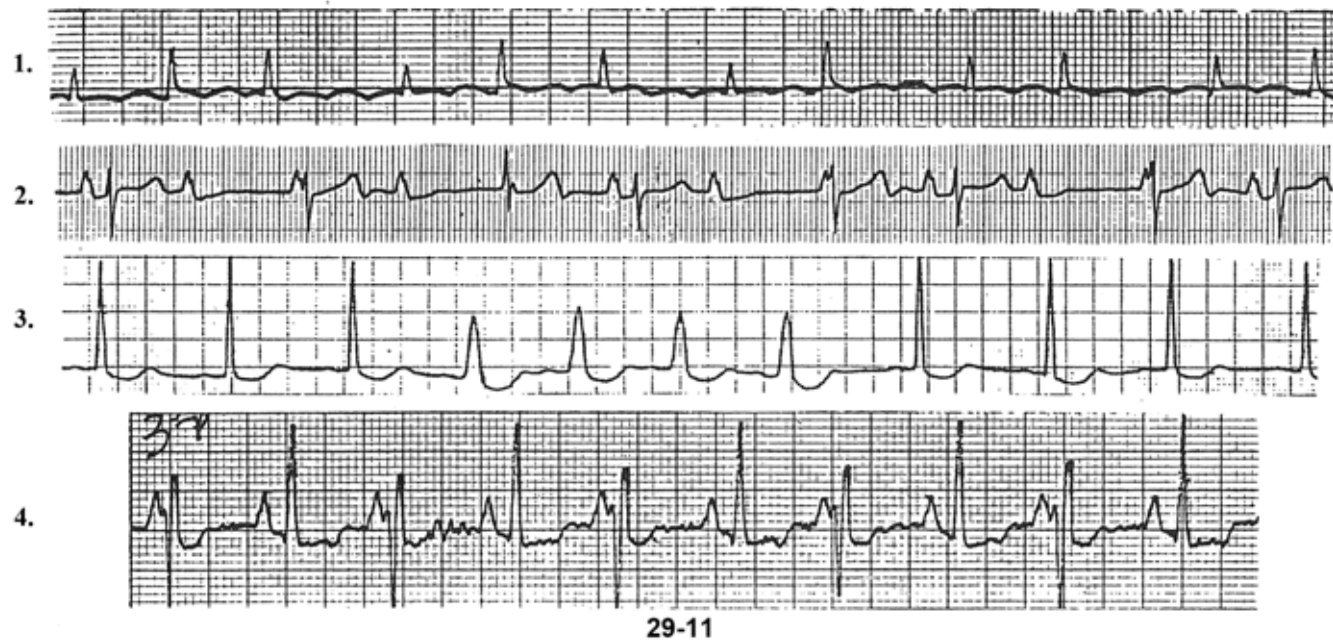
Tracing 1: During the pause, there is a negative blip soon after the QRS complex, which is a prematurely occurring P wave. That P wave occurs during the refractory period of the conduction system and is blocked, causing the pause. If the atrial premature impulse occurs a little later (sixth beat), when a part of the intraventricular conduction system (usually the left bundle branch) has recovered from the refractory period while the other part (usually the right bundle branch) is still refractory, it will conduct to the ventricles aberrantly, which is what is happening there. Thus, a premature atrial impulse can conduct to the ventricles normally, aberrantly or not conducted at all. In clinical practice, an unexpected pause is most often due to a nonconducted premature atrial impulse.

Tracing 2: P waves occur regularly. Some P waves are conducted to the ventricles while some others are blocked: 2° AV block. When they are conducting the P-R interval progressively lengthens. Hence, Type I.

Tracing 3: P waves occur regularly, some of which are conducted to the ventricles while others are blocked: 2° AV block. When they are conducting the P-R intervals do not lengthen: Type II.

Tracing 4: There are pauses in the midst of a regular rhythm. There are no extra P waves during the pauses, indicating that one is not dealing with AV block. The pause is exactly twice the shorter cycle length indicating regularly firing sinus impulses that fail to conduct to the atrium at times: SA block. Since the pause is twice the shorter cycle length, it is Type II. If it is less than twice the shorter cycle length, that means there is a progressive lengthening of SA conduction time before the block, indicating Type I. If the ventricular rate gradually slows down before the pause and gradually speeds up after the pause, the rhythm is sinus arrhythmia, not SA block. Thus, whether the change in R-R intervals occurs abruptly or gradually is an important observation to make.

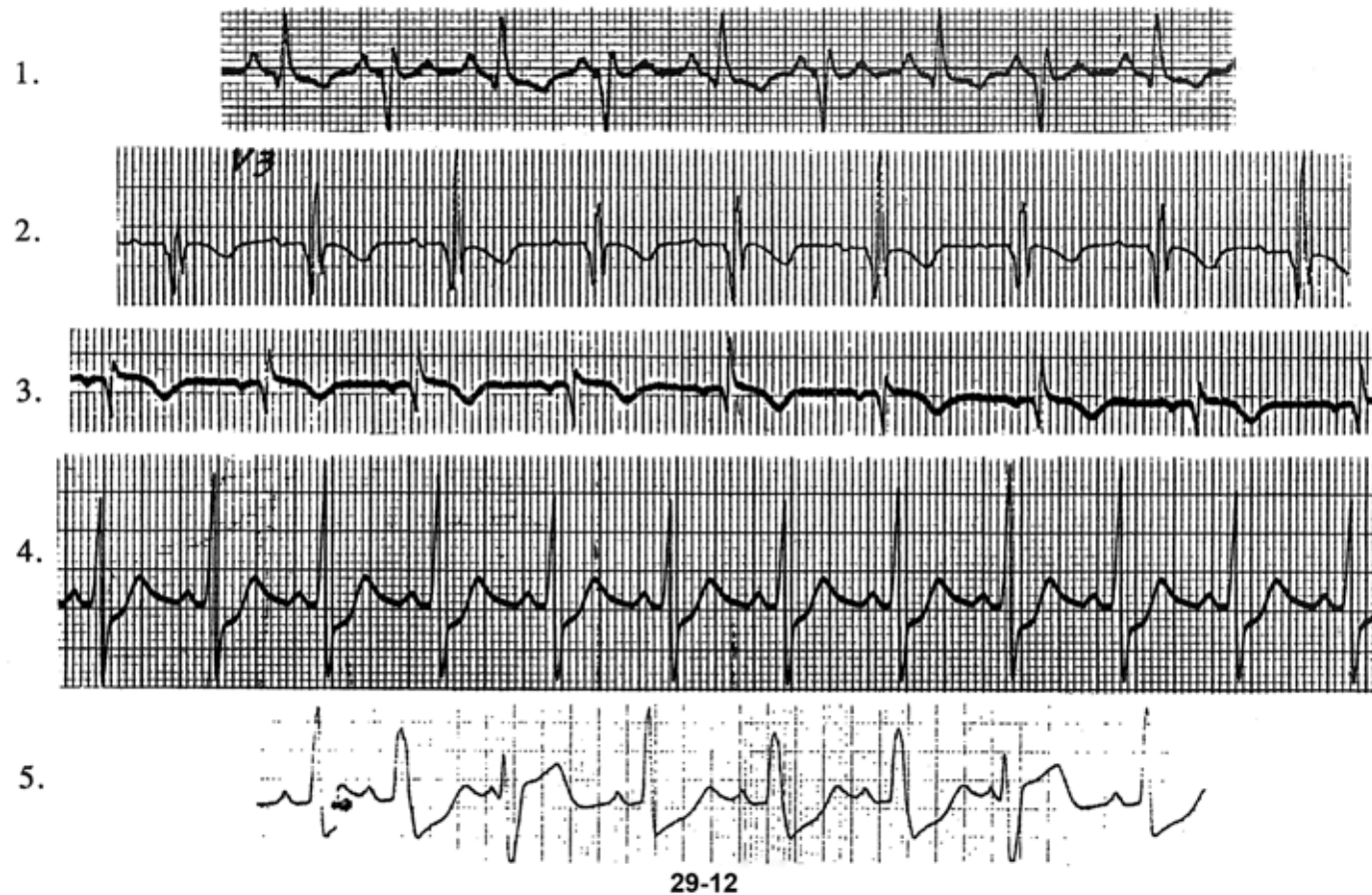
29-11 Question: What is the cause of changing QRS height or morphology?



Answer:

1. The changing QRS height is due to the timing of the QRS in relationship to the flutter waves. If a QRS occurs on the top of the “dome” of the flutter wave, then a taller R wave will result. If the QRS occurs in the “valley” of the flutter wave, the QRS height will be reduced.
2. The third QRS is different from any other QRS. This is due to the summation of the P wave and R wave judging from the timing of the P waves.
3. This is an example of a rate-dependent BBB. When the sinus rate speeds up, LBBB results. When the rate slows down, the conduction normalizes.
4. This is an example of ventricular bigeminy in which the PVCs occur late enough to allow the sinus P wave to be present in front of each PVC.

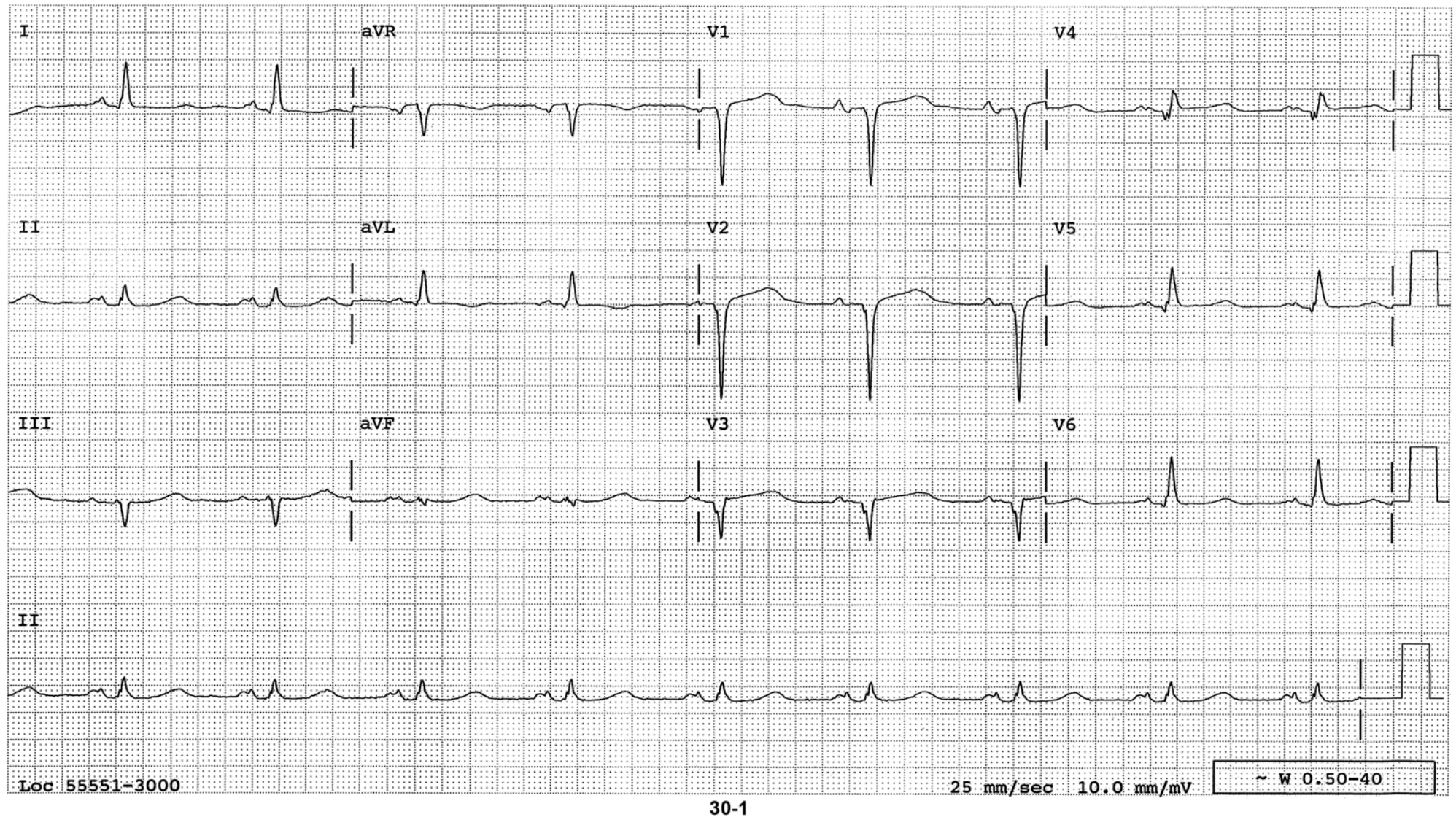
29-12 Question: What is the cause of changing QRS height or morphology?



Answer:

1. Ventricular bigeminy mimicking preexcitation of alternating beats. It is not electrical alternance (EA) because the R-R intervals are not the same. Short and long R-R intervals alternate.
2. Respiration causes the QRS height and configuration to change.
3. Another example of respiratory variation in QRS morphology mimicking EA in the latter part of the strip.
Note that two consecutive QRSs (the second and third QRSs) are more positive. In EA, they truly alternate. This is from a patient whose respiratory rate is one-half of the cardiac rate.
4. Respiratory variation resulting in a gradual increase and decrease in the height of the R wave.
5. Rate-dependent BBB. After the compensatory pause following a ventricular premature beat, the first sinus beat is most normally conducted.

SECTION 30



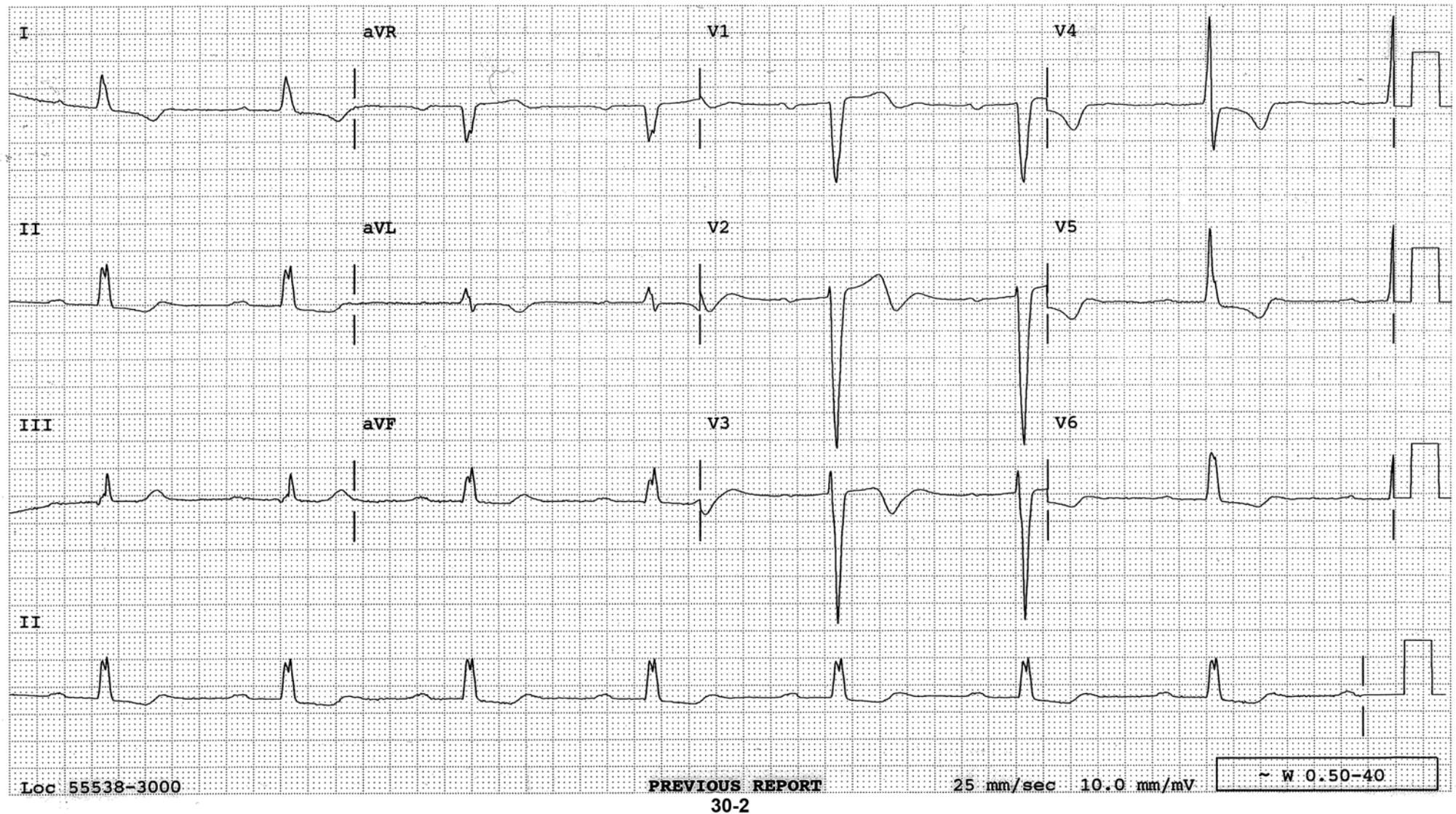
30-1 Sinus rhythm at a rate of 55/minute. The QS pattern in V_1 - V_3 , combined with a wide Q wave in V_4 is diagnostic of an old anterior infarct. The Q-T interval is markedly prolonged and measures about 600 milliseconds. This long Q-T interval predisposes the patient to *Torsade de Pointes*.

- Dx:
1. NSR
 2. Old anterior infarct
 3. Long Q-T interval

30-2 Question: This tracing reveals (choose one from below):

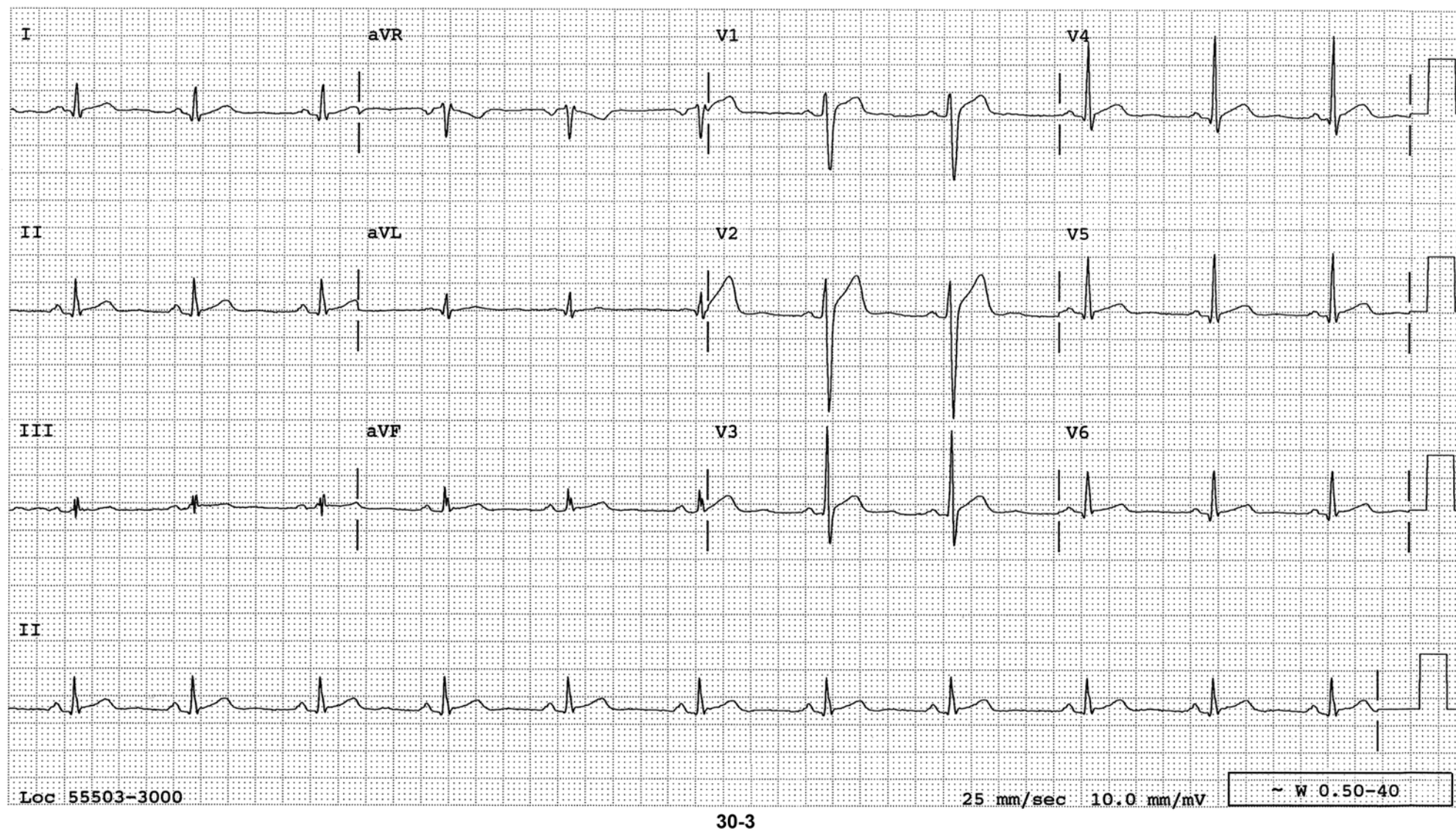
(A) Sinus bradycardia (HR around 40/minute), 1° AV block, IVCD and prolonged Q-T interval

(B) All the measurements (R-R interval, P-R interval, QRS duration and Q-T interval) are about twice the normal 2° to doubling of the paper speed.



Answer: (A) Sinus bradycardia (HR around 40/minute), 1° AV block, IVCD and prolonged Q-T interval

Clues: At first glance, the R-R interval, P-R interval, QRS duration and Q-T interval seem to be doubled, raising the possibility of doubling of the paper speed. If that were the case, the calibration mark at the end of the tracing should be wider (10 mm, not 5 mm), which is not the case here. Besides, at the bottom of the tracing, the actual paper speed is also printed and indicates "25 mm/second" which is the standard paper speed.

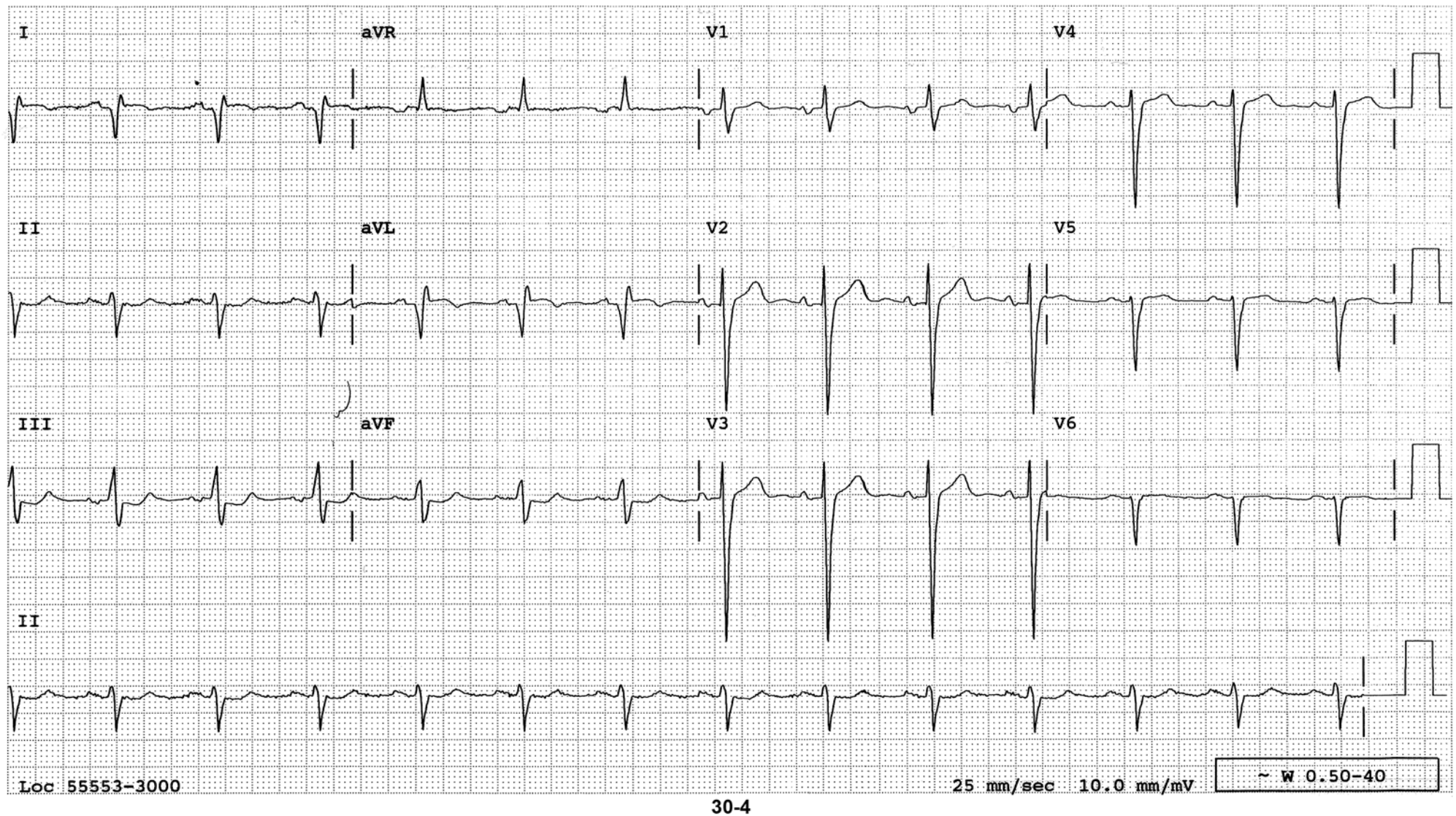


30-3 Normal sinus rhythm at a rate of 66/minute. The Q-T interval is short and measures 330 milliseconds. Digitalis or hypercalcemia can shorten the Q-T interval.

- Dx: 1. NSR
2. Short Q-T interval

30-4 Question: This tracing reveals (choose one from below):

- (A) Lateral MI
- (B) Reversed arm leads
- (C) Dextrocardia as a part of situs inversus
- (D) Lateral MI and right-sided precordial leads

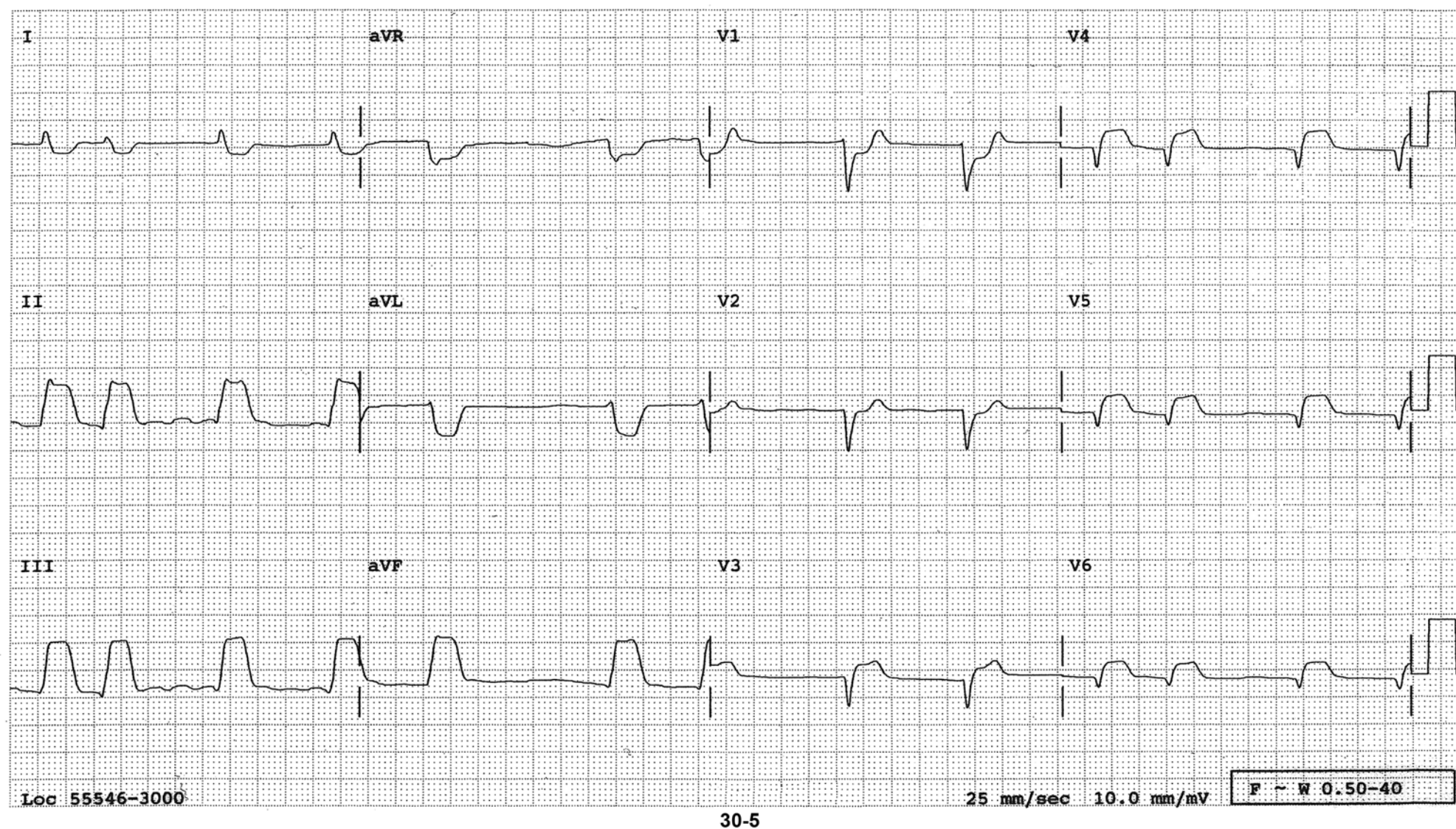


Answer: (A) Lateral MI

Discussion: Positive P waves in lead I and the left precordial leads immediately dispel the notion of reversed arm leads, dextrocardia, or right-sided precordial leads. Deep and wide Q waves in leads I and aVL are diagnostic of high lateral MI. Regressing R waves in V₄-V₆ are secondary to this lateral MI.

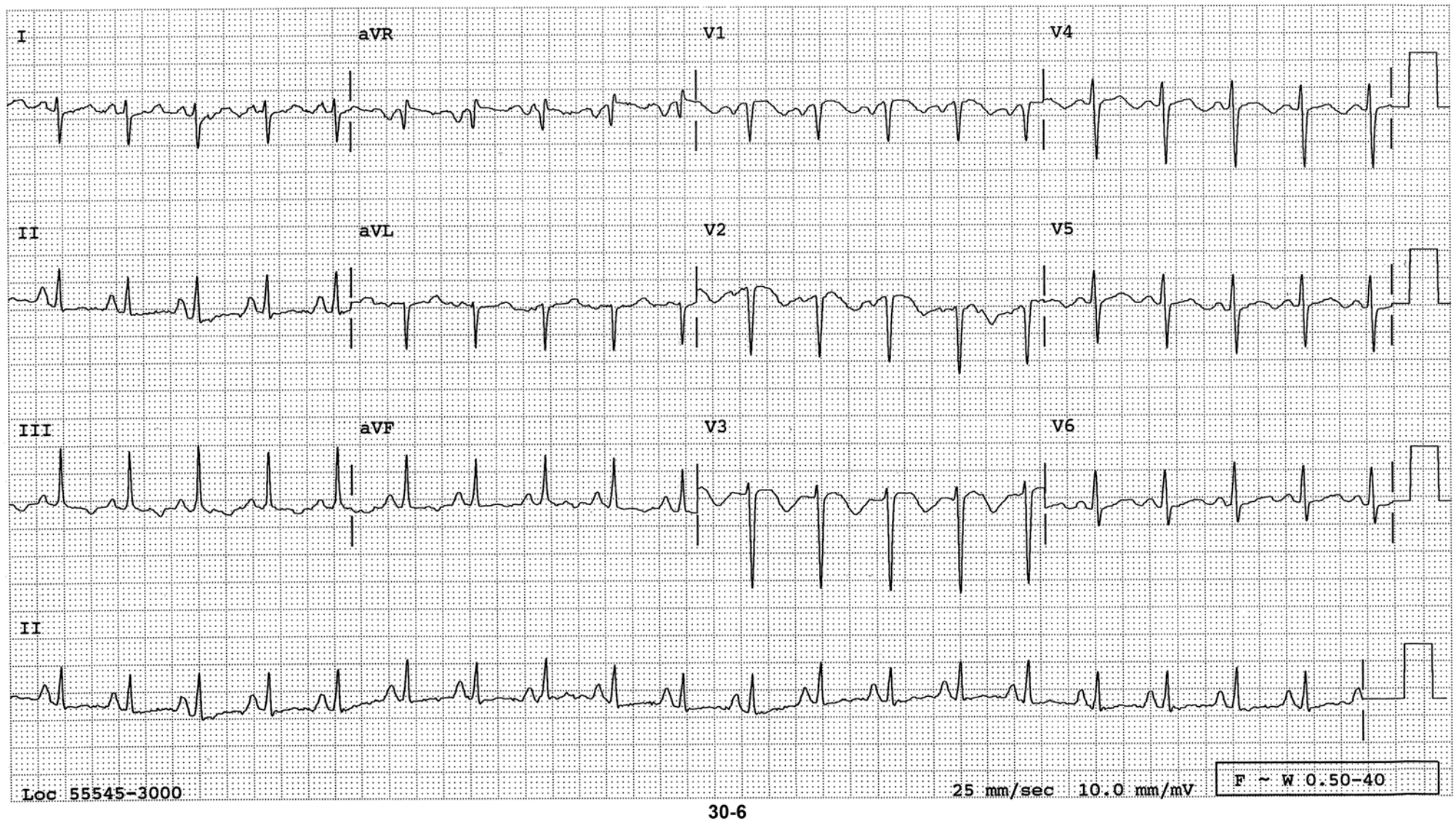
30.5 Question: This tracing reveals (choose one from below):

- (A) Atrial fib, acute inferoposterior infarct, and right-sided precordial leads revealing RV infarct
- (B) Atrial fib, acute inferior and anterior infarcts
- (C) Ventricular rhythm initially, then atrial fibrillation in the later half of the tracing and extensive anterior infarct



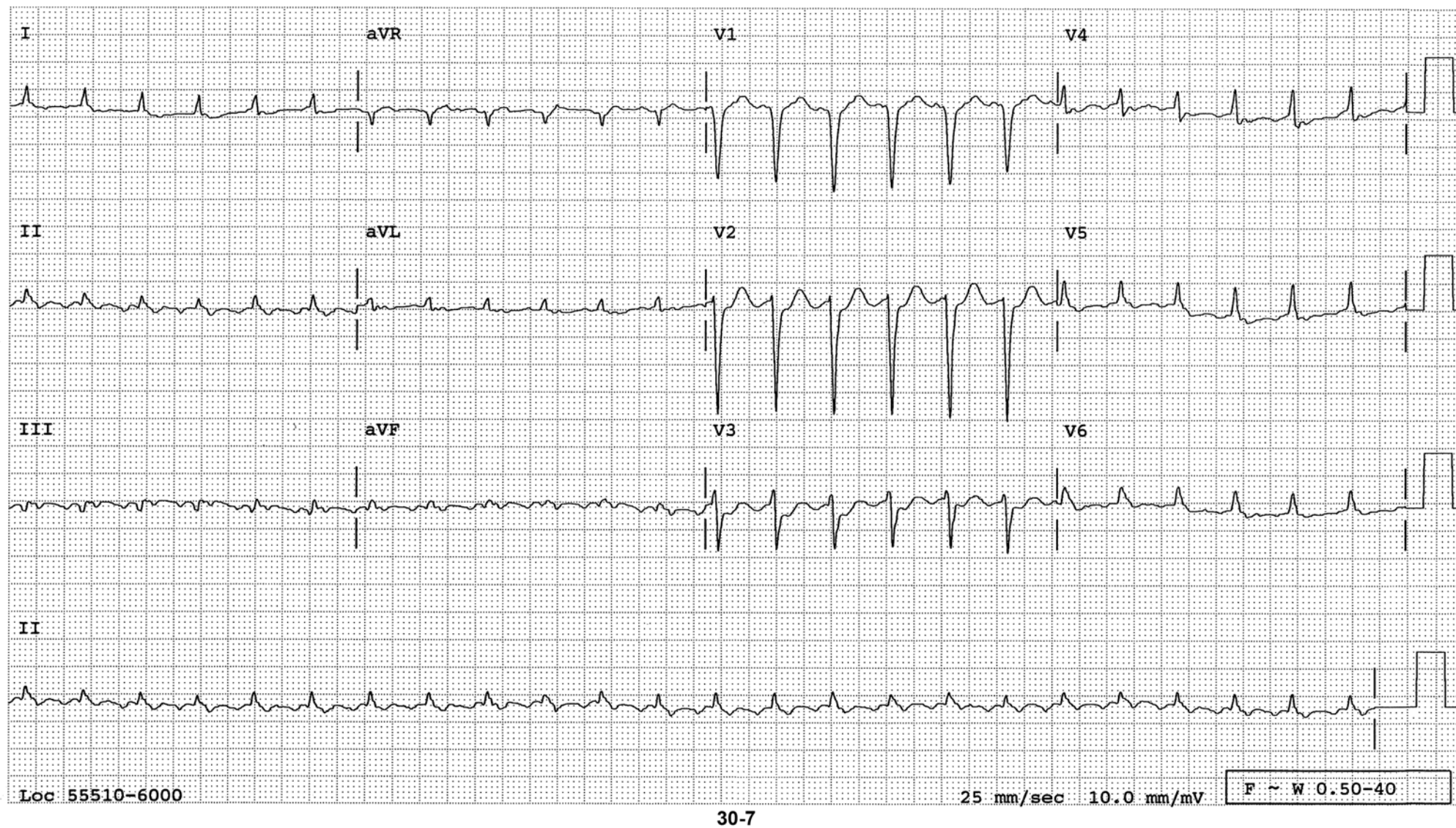
Correct Answer: (A) Atrial fibrillation, acute inferoposterior MI, right-sided precordial leads revealing RV involvement.

Clues: Leads I, aVL, and V_6 look at the heart from similar angles and should have a similar QRS morphology. In this case, the QRS vector in lead V_6 is opposite from those of leads I and aVL, indicating that the precordial leads are not regular left-sided but are actually right-sided. The ST elevation in the right-sided V_3 - V_6 are diagnostic of RV infarct. ST-segment is elevated as much as the height of the R wave in the inferior leads simulating a wide QRS complex, but it is from an inferior STEMI. In this patient, the lead V_1 is actually V_2 of regular left-sided precordial leads and horizontal ST depression there is due to the posterior wall involvement registered reciprocally.



30-6 Sinus tachycardia at a rate of 118/minute. RAD, S waves in the left precordial leads and T wave inversion in the right precordial leads are all diagnostic features of RVH. Since the R waves progress poorly in the right precordial leads, this RVH is due to COPD rather than other causes of RVH, such as primary pulmonary hypertension or other congenital heart diseases. The P wave is prominent in lead II, measuring 3 mm, and is indicative of RAE.

- Dx:*
1. Sinus tachycardia
 2. RAE
 3. RVH, most likely from COPD



30-7 Narrow QRS tachycardia at a rate of 143/minute. “Blunt sawtooth” pattern of atrial flutter is appreciable in the inferior leads, especially in the rhythm strip of lead II. The QRS voltage is abnormally low in the limb leads.

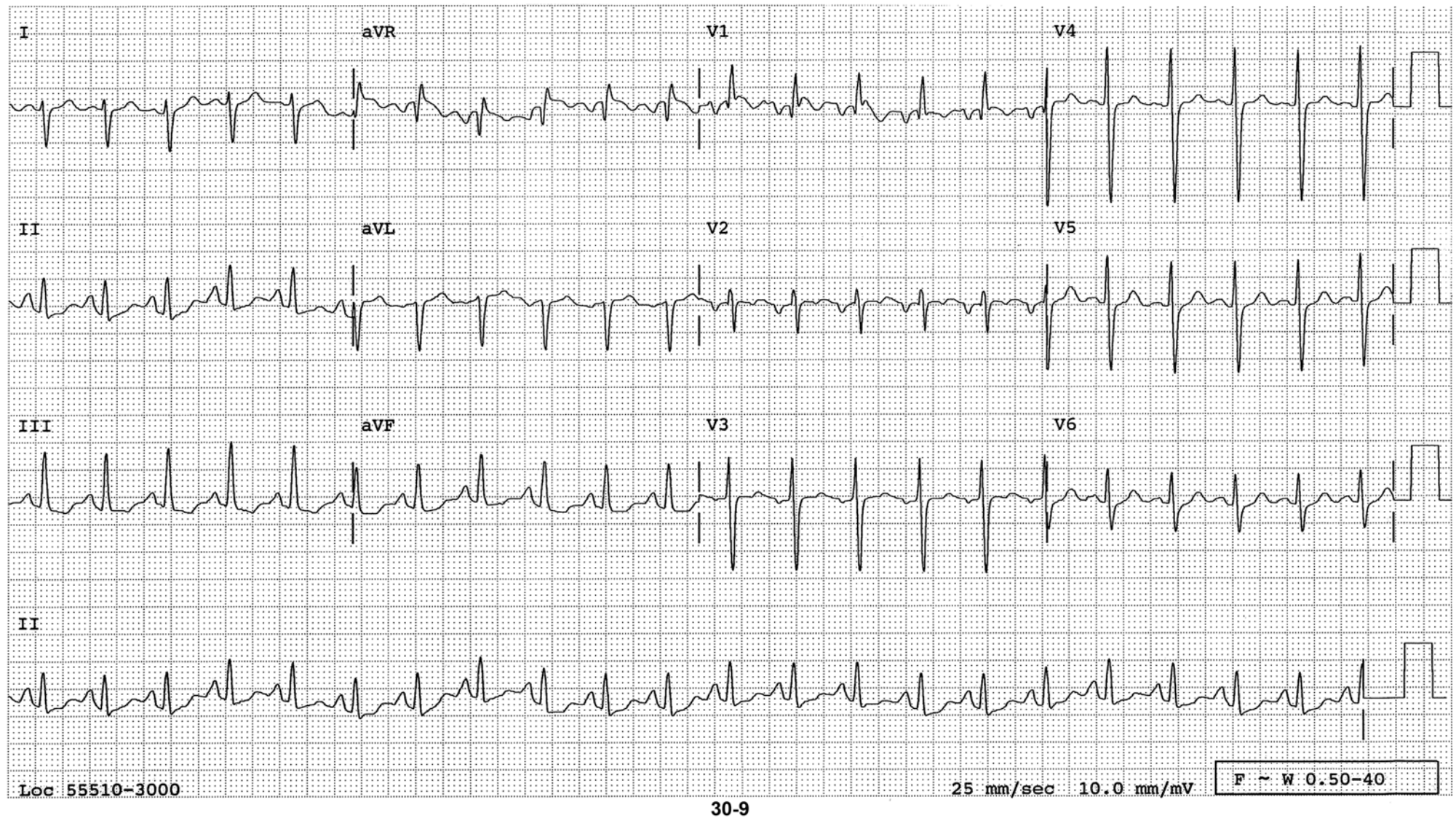
- Dx:*
1. Atrial flutter with 2:1 AV conduction
 2. Low QRS voltage in the limb leads
 3. Diffuse ST-T changes



30-8

- 30-8 These rhythm strips, taken at different times from the same patient, reveal intermittent 2° AV block. The P-R interval does not lengthen prior to the block, raising the possibility of Type II 2° AV block. However, the P to P interval significantly lengthens around the time when the P wave is blocked, suggesting that it is not an intrinsic AV node problem, but some other force extrinsic to the heart is affecting the sinus node to slow down as well as the AV node to block at the same time. That “some other force” is an increased vagal tone and is seen occasionally in well-conditioned athletes, and is benign. This tracing should not be interpreted as Mobitz Type II 2° AV block and implant a permanent pacemaker. When a P wave is blocked unexpectedly, paying attention to the P to P interval is useful as in this case.

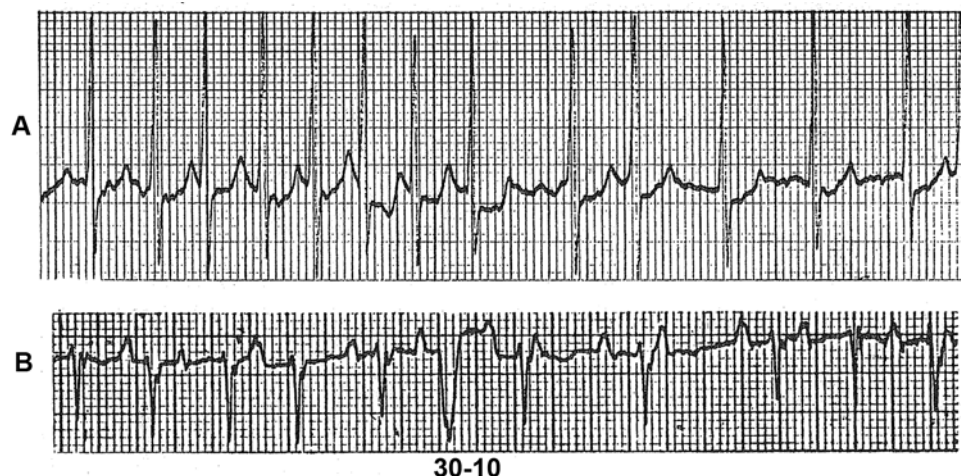
30-9 Question: What clinical condition should this ECG tracing make you think of?



Answer: Mitral stenosis

Discussion: RAD, tall R wave in V_1 - V_2 are all diagnostic features of RVH. Biphasic P wave with a prominent negative component in V_1 is good for LAE. The P wave is somewhat prominent in lead II suggesting right atrial enlargement as well.

In predominant mitral stenosis, the left ventricle is not affected, while the left atrium is enlarged, and varying degrees of RVH is present. Therefore, ECG findings of RVH and LAE in the absence of LVH should make one think of predominant mitral stenosis.



30-10 These two tracings from two different patients have similarities and dissimilarities. The similarities are:

- (A) Irregularly irregular rhythm
- (B) Pointed blips suggestive of atrial activities

Discussion: In tracing A, the pointed blips have a fixed relationship to the preceding QRS, and these are T waves, and the underlying rhythm is atrial fibrillation. In tracing B, the pointed blips are P waves and this tracing is an example of MAT. Note that the P-P intervals are changing, the P wave morphology is changing, and the P-R and R-R intervals are changing. These blips do not have a fixed relationship to the preceding QRS as in tracing A. The R-P and P-R intervals have a reciprocal relationship, i.e. if the P wave occurs relatively away from the preceding QRS, the AV junction will have more time to recover, and the P-R interval will be shorter. When the P wave occurs relatively closer to the preceding QRS, the AV junctional tissue is still partially refractory and it will result in a longer P-R interval. If the P wave occurs too early, it will not be conducted to the ventricle at all, resulting in a nonconducted P wave.

30-11 *Question:* Match the tracings from patient A and patient B with the diagnoses listed below:

(A) Mobitz Type I 2° AV block

(B) Mobitz Type II 2° AV block



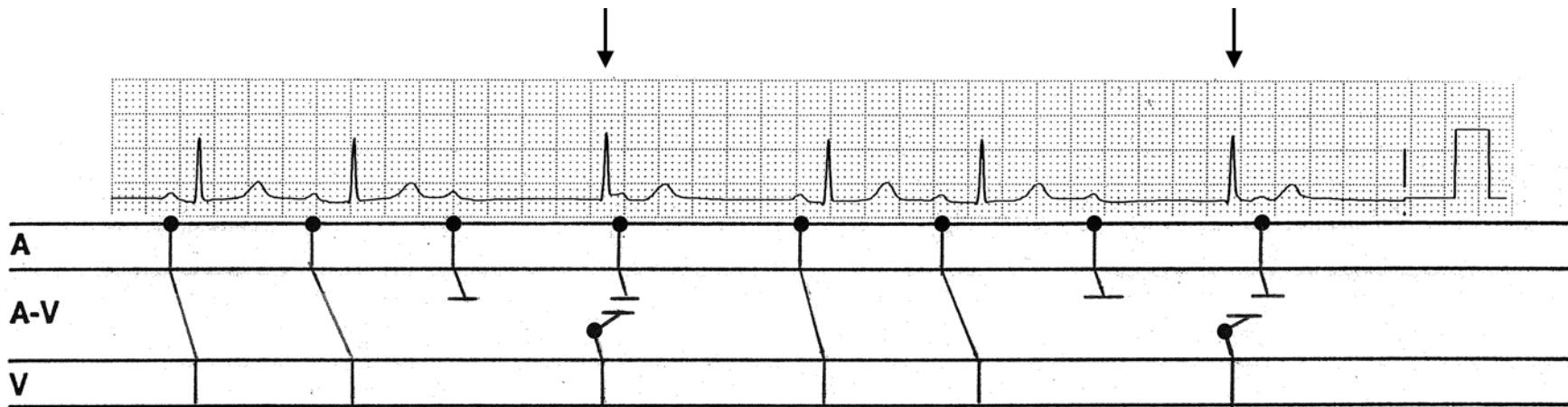
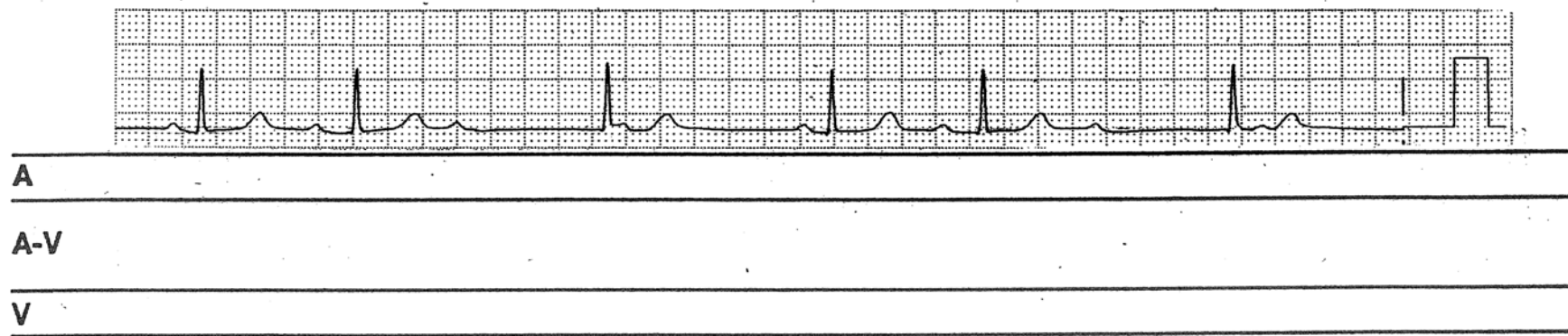
Answer: Patient A has Mobitz Type II 2° AV block.

The AV conduction ratio is 3:1 at the beginning and 2:1 subsequently. Two or more consecutive P waves not conducting and wide QRS complexes are good features of Type II 2° AV block.

Patient B has Mobitz type I 2° AV block

The tracing in patient B reveals Type I 2° AV block with 3:2 AV conduction ratio initially, then 2:1 AV block in the middle, and then back to 3:2 AV conduction toward the end of the strip. One should not consider this as Type I and Type II 2° AV block coexisting in the same patient. This patient merely has Type I 2° AV block with a problem within the AV node due to reversible conditions. Wenckebach phenomenon can progress from, say, 5:4 to 4:3, then to 3:2, and the next step would be 2:1 AV block. Thus, both Type I and Type II 2° AV block can result in 2:1 AV block. If there is any Wenckebach periodicity elsewhere in that tracing, that 2:1 AV block is part of Type I.

Please draw the ladder diagram in the space provided below.



30-12

30-12

(Footnote)

The primary conduction problem in this tracing is AV Wenckebach phenomenon (Type I 2° AV block). After the blocked P wave, the pause is too long and allows the AV junctional pacemaker to escape (↓).

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